```
      (FILE 'HOME' ENTERED AT 16:10:26 ON 05 APR 2007)

      INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
      AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
      CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
      DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 16:10:40 ON 05 APR 2007
                SEA (LYSOPHOSPHATIDIC(W)ACID)AND(ATHEROSCLEROSIS OR NEOINTIMA)A
                ---------
                 7   FILE BIOSIS
                10   FILE CAPLUS
                 1   FILE DDFU
                 6   FILE DGENE
                 1   FILE DRUGU
                10   FILE EMBASE
                 3   FILE ESBIOBASE
                 1   FILE IFIPAT
                 1   FILE LIFESCI
                 4   FILE MEDLINE
                 5   FILE PROUSDDR
                 7   FILE SCISEARCH
                 4   FILE TOXCENTER
               310   FILE USPATFULL
                45   FILE USPAT2
                 9   FILE WPIDS
                 9   FILE WPINDEX
L1               QUE (LYSOPHOSPHATIDIC(W) ACID) AND(ATHEROSCLEROSIS OR NEOINTIMA
                 ---------

      FILE 'EMBASE, CAPLUS' ENTERED AT 16:12:09 ON 05 APR 2007
L2            20 S (LYSOPHOSPHATIDIC(W)ACID)AND(ATHEROSCLEROSIS OR NEOINTIMA)AND
L3            16 DUP REM L2 (4 DUPLICATES REMOVED)
L4             9 S L3 NOT PY>2004
L5             0 S TIGYI/AU
L6             0 S TIGYI, GREGOR/AU

      FILE 'CAPLUS' ENTERED AT 16:32:03 ON 05 APR 2007
L7          2449 S LYSOPHOSPHATIDIC(W)ACID
L8            98 S L7 AND (ATHEROSCLEROSIS OR NEOINTIMA OR CARDIOVASCULAR)
L9            50 S L8 AND (ANTAGON? OR INHIB?)
L10           27 S L9 NOT PY>2004
L11           24 S L10 NOT L4
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=> file registry
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 16:46:43 ON 23 MAR 2007
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 22 MAR 2007 HIGHEST RN 927959-98-6
DICTIONARY FILE UPDATES: 22 MAR 2007 HIGHEST RN 927959-98-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

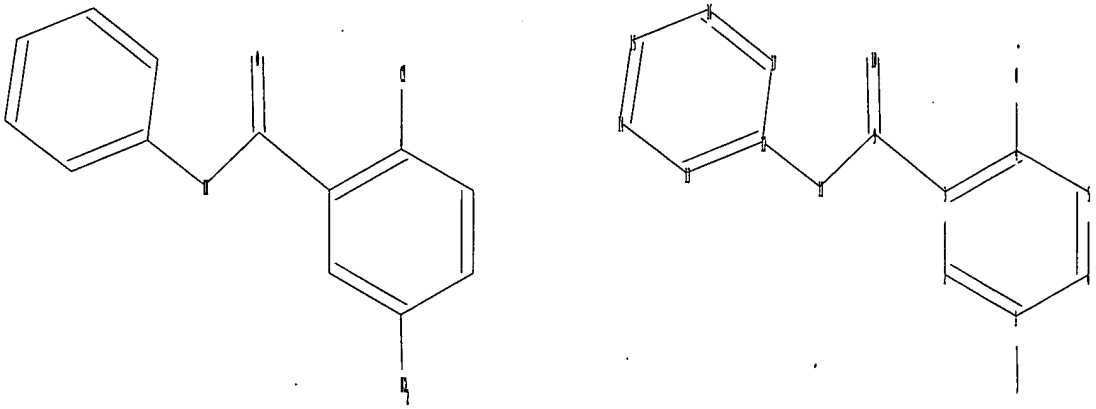
TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=>
Uploading C:\Program Files\Stnexp\Queries\10821739GW9662.str

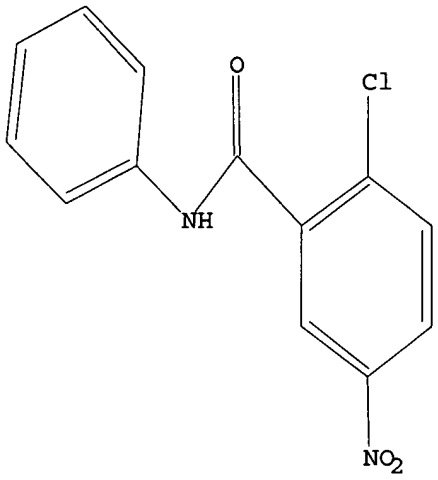


chain nodes :
7 8 9 10 11
ring nodes :
1 2 3 4 5 6 12 13 14 15 16 17
chain bonds :
1-7 3-9 4-8 9-10 9-11 11-12
ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6 12-13 12-17 13-14 14-15 15-16 16-17
exact/norm bonds :
9-10 9-11 11-12
exact bonds :
1-7 3-9 4-8
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6 12-13 12-17 13-14 14-15 15-16 16-17

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:CLASS 8:CLASS 9:CLASS 10:CLASS
11:CLASS 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom

L1 STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1 STR



Structure attributes must be viewed using STN Express query preparation.

=> s l1
SAMPLE SEARCH INITIATED 16:47:05 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 138 TO ITERATE

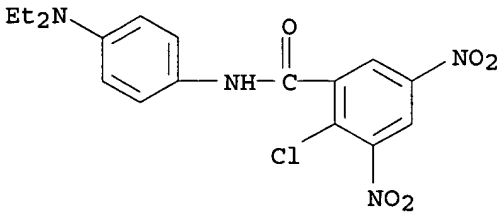
100.0% PROCESSED 138 ITERATIONS 50 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 2056 TO 3464
PROJECTED ANSWERS: 981 TO 2019

L2 50 SEA SSS SAM L1

=> d l2 scan

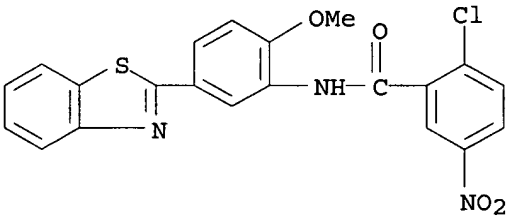
L2 50 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN INDEX NAME NOT YET ASSIGNED
MF C17 H17 Cl N4 O5



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

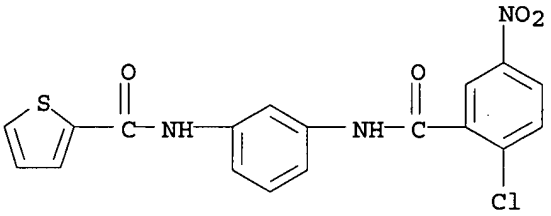
HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):3

L2 50 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN INDEX NAME NOT YET ASSIGNED
MF C21 H14 Cl N3 O4 S



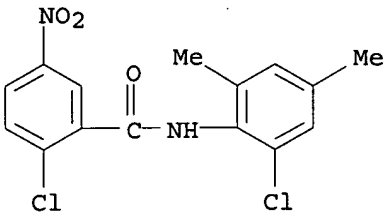
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L2 50 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN 2-Thiophenecarboxamide, N-[3-[(2-chloro-5-nitrobenzoyl)amino]phenyl]- (9CI)
MF C18 H12 Cl N3 O4 S



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L2 50 ANSWERS REGISTRY COPYRIGHT 2007 ACS on STN
IN Benzamide, 2-chloro-N-(2-chloro-4,6-dimethylphenyl)-5-nitro- (9CI)
MF C15 H12 Cl2 N2 O3



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s l1 fam sam
SAMPLE SEARCH INITIATED 16:47:25 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 5 TO ITERATE

100.0% PROCESSED 5 ITERATIONS 0 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 5 TO 234
PROJECTED ANSWERS: 0 TO 0

L3 0 SEA FAM SAM L1

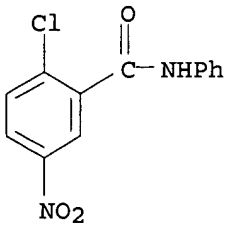
=> s l1 fam full
FULL SEARCH INITIATED 16:47:30 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 116 TO ITERATE

100.0% PROCESSED 116 ITERATIONS 1 ANSWERS
SEARCH TIME: 00.00.01

L4 1 SEA FAM FUL L1

=> d l4

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
RN 22978-25-2 REGISTRY
ED Entered STN: 16 Nov 1984
CN Benzamide, 2-chloro-5-nitro-N-phenyl- (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzanilide, 2-chloro-5-nitro- (8CI)
OTHER NAMES:
CN 2-Chloro-5-nitrobenzanilide
CN GW 9662
MF C13 H9 Cl N2 O3
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CSCHEM, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

56 REFERENCES IN FILE CA (1907 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
58 REFERENCES IN FILE CAPLUS (1907 TO DATE)

```
=> file caplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      70.10      70.31

FILE 'CAPLUS' ENTERED AT 16:47:41 ON 23 MAR 2007
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FILE COVERS 1907 - 23 Mar 2007  VOL 146 ISS 14
FILE LAST UPDATED: 22 Mar 2007  (20070322/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l4
L5          58 L4

=> s l4/thu
            58 L4
        870194 THU/RL
L6          21 L4/THU
                (L4 (L) THU/RL)

=> s l5 and neointima
          1779 NEOINTIMA
L7           1 L5 AND NEOINTIMA

=> d l7 ti abs bib

L7   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
```

TI Lysophosphatidic acid analogs and inhibition of neointima formation

AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPARγ, abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPARγ. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPARγ or antagonists of PPARγ that inhibit PPARγ signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.

AN 2004:857161 CAPLUS <<LOGINID::20070323>>

DN 141:343506

TI Lysophosphatidic acid analogs and inhibition of neointima formation

IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang

PA USA

SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004204383	A1	20041014	US 2004-821739	20040409
	AU 2004229467	A1	20041028	AU 2004-229467	20040409
	CA 2521189	A1	20041028	CA 2004-2521189	20040409
	WO 2004091496	A2	20041028	WO 2004-US11016	20040409
	WO 2004091496	A3	20050324		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1613298	A2	20060111	EP 2004-759365	20040409
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
PRAI	US 2003-462274P	P	20030411		
	WO 2004-US11016	W	20040409		

=> s l5 and atherosclerosis
53556 ATHEROSCLEROSIS
L8 3 L5 AND ATHEROSCLEROSIS

=> d l8 1-3 ti

L8 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Nitrated lipids and methods of making and using thereof

L8 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation

L8 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Thiazolidinediones, a class of anti-diabetic drugs, inhibit Id2 expression through a PPARγ-independent pathway in human aortic smooth muscle cells

=> d l8 1 3 ti abs bib

L8 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Nitrated lipids and methods of making and using thereof
AB Described herein are nitrated lipids and methods of making and using the nitrated lipids.
AN 2005:1239024 CAPLUS <<LOGINID::20070323>>
DN 144:601
TI Nitrated lipids and methods of making and using thereof
IN Freeman, Bruce A.; Schopfer, Francisco; O'Donnell, Valerie; Baker, Paul; Chen, Eugene; Branchaud, Bruce
PA Uab Research Foundation, USA
SO PCT Int. Appl., 169 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005110396	A2	20051124	WO 2005-US14305	20050426
	WO 2005110396	A3	20070301		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2004-566005P P 20040428
OS MARPAT 144:601

L8 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
TI Thiazolidinediones, a class of anti-diabetic drugs, inhibit Id2 expression through a PPARγ-independent pathway in human aortic smooth muscle cells
AB Inhibitor of DNA binding (Id2) is a member of the helix-loop-helix family of transcription regulators that is known to play important roles in the proliferation and differentiation of many cell types. Overexpression of Id2 has been reported to result in significant enhancement of vascular smooth muscle cell growth via increased S phase entry. We hypothesized that downregulation of Id2 gene expression by thiazolidinediones (TZDs), a class of anti-diabetic drugs and peroxisome proliferator-activated receptor γ (PPARγ) activators, might contribute to the anti-atherosclerotic and anti-hypertensive effects of the PPARγ. Here we document that TZDs, including troglitazone and ciglitazone, repress Id2 gene expression in a doses- and time-dependent manner. However, GW7845, a high-affinity and non-TZD PPARγ activator, had no inhibitory effect on Id2 gene expression. In addition, PPARγ antagonist GW9662 did not rescue TZD-induced Id2 repression. Taken together, our data suggest that TZDs repress Id2 expression through a PPARγ-independent pathway.
AN 2003:199053 CAPLUS <<LOGINID::20070323>>

DN 139:78835
TI Thiazolidinediones, a class of anti-diabetic drugs, inhibit Id2 expression through a PPARγ-independent pathway in human aortic smooth muscle cells
AU Zhu, X.; Lin, Y.; Zhang, J.; Fu, M.; Mao, Z.; Chen, Y. E.
CS Cardiovascular Research Institute, Peking University Health Science Center, Beijing, 100083, Peop. Rep. China
SO Cellular and Molecular Life Sciences (2003), 60(1), 212-218
CODEN: CMLSFI; ISSN: 1420-682X
PB Birkhaeuser Verlag
DT Journal
LA English
RE.CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l5 and (PPAR or (peroxisome(w)proliferator-activated(w)gamma))
8826 PPAR
17707 PEROXISOME
11527 PROLIFERATOR
518979 ACTIVATED
10513 PROLIFERATOR-ACTIVATED
(PROLIFERATOR(W)ACTIVATED)
843860 GAMMA
17 PEROXISOME(W)PROLIFERATOR-ACTIVATED(W)GAMMA
L9 40 L5 AND (PPAR OR (PEROXISOME(W)PROLIFERATOR-ACTIVATED(W)GAMMA))

=> s l9 not py>2004
2882076 PY>2004
L10 11 L9 NOT PY>2004

=> d l10 1-11 ti

L10 ANSWER 1 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Genistein inhibits expressions of NADPH oxidase p22phox and angiotensin II type 1 receptor in aortic endothelial cells from stroke-prone spontaneously hypertensive rats

L10 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI GW9662, a potent antagonist of PPAR.gamma., inhibits growth of breast tumour cells and promotes the anticancer effects of the PPAR.gamma. agonist rosiglitazone, independently of PPAR γ activation

L10 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Antidiabetic thiazolidinediones inhibit invasiveness of pancreatic cancer cells via PPAR.gamma. independent mechanisms

L10 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Inhibition of cell proliferation by potential peroxisome proliferator-activated receptor (PPAR) gammaa agonists and antagonists

L10 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Troglitazone acts on cellular pH and DNA synthesis through a peroxisome proliferator-activated receptor γ-independent mechanism in breast cancer-derived cell lines

L10 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Angiotensin Type 1 Receptor Blockers Induce Peroxisome Proliferator-Activated Receptor-γ Activity

L10 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Binding analyses between human PPAR.gamma.-LBD and ligands:

surface plasmon resonance biosensor assay correlating with circular dichroic spectroscopy determination and molecular docking

L10 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Rosiglitazone, a ligand of the peroxisome proliferator-activated receptor-γ, reduces the development of nonseptic shock induced by zymosan in mice

L10 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Thiazolidinediones, a class of anti-diabetic drugs, inhibit Id2 expression through a PPAR.gamma.-independent pathway in human aortic smooth muscle cells

L10 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Functional Consequences of Cysteine Modification in the Ligand Binding Sites of Peroxisome Proliferator Activated Receptors by GW9662

L10 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Novel nitroaryl amides as nuclear receptor arylating compounds

=> d l10 2 3 4 5 8 9 10 11 ti abs bib

L10 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI GW9662, a potent antagonist of PPAR.gamma., inhibits growth of breast tumour cells and promotes the anticancer effects of the PPAR.gamma. agonist rosiglitazone, independently of PPAR γ activation
AB Peroxisome proliferator-activated receptor gamma (PPAR.gamma.), a member of the nuclear receptor superfamily, is activated by several compds., including the thiazolidinediones. In addition to being a therapeutic target for obesity, hypolipidemia and diabetes, perturbation of PPAR.gamma. signaling is now believed to be a strategy for treatment of several cancers, including breast. Although differential expression of PPAR.gamma. is observed in tumors compared to normal tissues and PPAR.gamma. agonists have been shown to inhibit tumor cell growth and survival, the interdependence of these observations is unclear. This study demonstrated that the potent, irreversible and selective PPAR.gamma. antagonist GW9662 prevented activation of PPAR.gamma. and inhibited growth of human mammary tumor cell lines. Controversially, GW9662 prevented rosiglitazone-mediated PPAR.gamma. activation, but enhanced rather than reversed rosiglitazone-induced growth inhibition. As such, these data support the existence of PPAR.gamma.-independent pathways and question the central belief that PPAR.gamma. ligands mediate their anticancer effects via activation of PPAR.gamma..
AN 2005:19155 CAPLUS <<LOGINID::20070323>>
DN 142:126885
TI GW9662, a potent antagonist of PPAR.gamma., inhibits growth of breast tumour cells and promotes the anticancer effects of the PPAR.gamma. agonist rosiglitazone, independently of PPAR γ activation
AU Seargent, Jill M.; Yates, Elisabeth A.; Gill, Jason H.
CS Cancer Research Unit, Tom Connor's Cancer Research Centre, University of Bradford, Bradford, BD7 1DP, UK
SO British Journal of Pharmacology (2004), 143(8), 933-937
CODEN: BJPCBM; ISSN: 0007-1188
PB Nature Publishing Group
DT Journal
LA English
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

TI Antidiabetic thiazolidinediones inhibit invasiveness of pancreatic cancer cells via PPAR.gamma. independent mechanisms

AB Thiazolidinediones (TZD) are a new class of oral antidiabetic drugs that have been shown to inhibit growth of some epithelial cancer cells. Although TZD were found to be ligands for peroxisome proliferators activated receptor γ (PPAR.gamma.) the mechanism by which TZD exert their anticancer effect is currently unclear. Furthermore, the effect of TZD on local motility and metastatic potential of cancer cells is unknown. The authors analyzed the effects of two TZD, rosiglitazone and pioglitazone, on invasiveness of human pancreatic carcinoma cell lines in order to evaluate the potential therapeutic use of these drugs in pancreatic adenocarcinoma. Expression of PPAR.gamma. in human pancreatic adenocarcinomas and pancreatic carcinoma cell lines was measured by reverse transcription polymerase chain reaction and confirmed by western blot anal. PPAR.gamma. activity was evaluated by transient reporter gene assay. Invasion assay was performed in modified Boyden chambers. Gelatinolytic and fibrinolytic activity were evaluated by gel zymog. TZD inhibited pancreatic cancer cells' invasiveness, affecting gelatinolytic and fibrinolytic activity with a mechanism independent of PPAR.gamma. activation and involving MMP-2 and PAI-1 expression. TZD treatment in pancreatic cancer cells has potent inhibitory effects on growth and invasiveness suggesting that these drugs may have application for prevention and treatment of pancreatic cancer in humans.

AN 2004:1020460 CAPLUS <<LOGINID::20070323>>
DN 142:232623

TI Antidiabetic thiazolidinediones inhibit invasiveness of pancreatic cancer cells via PPAR.gamma. independent mechanisms

AU Galli, A.; Ceni, E.; Crabb, D. W.; Mello, T.; Salzano, R.; Grappone, C.; Milani, S.; Surrenti, E.; Surrenti, C.; Casini, A.
CS Gastroenterology Unit, Department of Clinical Pathophysiology, University of Florence, Florence, Italy

SO Gut (2004), 53(11), 1688-1697
CODEN: GUTTAK; ISSN: 0017-5749
PB BMJ Publishing Group
DT Journal
LA English
RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN

TI Inhibition of cell proliferation by potential peroxisome proliferator-activated receptor (PPAR) gammaa agonists and antagonists

AB This study was initiated to determine if potential PPAR gamma antagonists could block the inhibition of cell proliferation caused by 4 phenylbutyrate. The action of 4 phenylbutyrate differed from other PPAR gamma ligands examined in that it induces histone acetylation. Proliferation of DS19 mouse erythroleukemia cells was inhibited by PPAR gamma agonists (4 phenylbutyrate, rosiglitazone, ciglitazone and GW1929) and by potential PPAR gamma antagonists: BADGE (Biphenol A diglycidyl ether), GW9662, PD068235 and diclofenac. Combined incubations tended to exhibit additive inhibitory effects. Potential PPAR gamma agonists and antagonists inhibited the incorporation of thymidine into DNA of human prostate (PC3), colon (Caco-2) and breast (T47D) cancer cells but also affected NIH3T3 cells that have little or no expression of PPAR gamma. Lipid accumulation in T47D cells was seen after incubation with 4 phenylbutyrate and both potential PPAR gamma agonists and antagonists. The extent to which the effects of 4 phenylbutyrate on cell proliferation are mediated through PPAR gamma or induction of histone acetylation remains an open question. We conclude that potential PPAR gamma antagonists may fail to reverse the growth inhibitory effect of PPAR gamma ligands and may themselves act as growth inhibitory agents.

AN 2004:1000244 CAPLUS <<LOGINID::20070323>>
DN 142:190496
TI Inhibition of cell proliferation by potential peroxisome proliferator-activated receptor (PPAR) gammaa agonists and antagonists
AU Lea, Michael A.; Sura, Monali; Desbordes, Charles
CS Department of Biochemistry and Molecular Biology, UMDNJ-New Jersey Medical School, Newark, NJ, 07103, USA
SO Anticancer Research (2004), 24(5A), 2765-2771
CODEN: ANTRD4; ISSN: 0250-7005
PB International Institute of Anticancer Research
DT Journal
LA English
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Troglitazone acts on cellular pH and DNA synthesis through a peroxisome proliferator-activated receptor γ-independent mechanism in breast cancer-derived cell lines
AB The purpose of this study was to assess whether troglitazone (TRO) would induce cellular acidosis by inhibiting Na+/H+ exchanger (NHE) 1 in breast carcinoma-derived cell lines and, if so, whether cellular acidosis would be associated with a reduction in proliferation. Intracellular pH (pHi) and acid extrusion capacity after an exogenous acid load were assayed using (2, 7)-biscarboxyethyl-5(6)-carboxyfluorescein in MCF-7 and MDA-MB-231 cells treated with TRO. Radiolabeled thymidine incorporation was used to assess DNA synthesis. Peroxisome proliferator-activated receptor (PPAR) γ involvement was assessed using an antagonist and PPAR γ-/- NIH3T3 cells. TRO induced a prompt (<4 min) and severe cellular acidosis in both MCF-7 (7.54 ± 0.23 to 6.77 ± 0.06; $P < 0.001$) and MDA-MB-231 cells (7.38 ± 0.18 to 6.89 ± 0.25; $P < 0.05$) after 12 min, without increasing acid production Acid extrusion as assessed by the response to an exogenous acid load (NH4Cl pulse) was markedly blunted (MDA-MB-231, $P < 0.01$) or eliminated (MCF-7, $P < 0.001$). Chronic exposure to TRO resulted in NHE1 activity reduction ($P < 0.05$) and a dose-dependent decrease in DNA synthesis (<75% inhibition at 100 μmol/L; $P < 0.001$ and $P < 0.01$ for MCF-7 and MDA-MB-231, resp.) associated with a decreased number of viable cells. TRO-mediated inhibition of proliferation was not reversed by the presence of the PPAR γ inhibitor GW9662 and was demonstrable in PPAR.gamma.-/- NIH3T3 cells, consistent with a PPAR.gamma.-independent mechanism. TRO induces marked cellular acidosis in MCF-7 and MDA-MD-231 cells. Sustained acidosis is consonant with decreased proliferation and growth that is not reversed by a PPAR.gamma. antagonist. Our results support a NHE-mediated action of TRO that exerts its effect independent of PPAR.gamma..
AN 2004:975687 CAPLUS <<LOGINID::20070323>>
DN 142:190487
TI Troglitazone acts on cellular pH and DNA synthesis through a peroxisome proliferator-activated receptor γ-independent mechanism in breast cancer-derived cell lines
AU Turturro, Francesco; Friday, Ellen; Fowler, Rocky; Surie, Diya; Welbourne, Tomas
CS Department of Medicine, Feist-Weiller Cancer Center, Louisiana State University Health Sciences Center, Shreveport, LA, USA
SO Clinical Cancer Research (2004), 10(20), 7022-7030
CODEN: CCREF4; ISSN: 1078-0432
PB American Association for Cancer Research
DT Journal
LA English
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Rosiglitazone, a ligand of the peroxisome proliferator-activated receptor-γ, reduces the development of nonseptic shock induced by zymosan in mice
AB OBJECTIVE: Peroxisome proliferator-activated receptors (PPARs) are members of the nuclear hormone receptor superfamily of ligand-activated transcription factors that are related to retinoid, steroid, and thyroid hormone receptors. The PPAR-γ receptor subtype appears to play a pivotal role in the regulation of cellular proliferation and inflammation. Rosiglitazone (Avandia) is a PPAR-γ agonist (the most potent PPAR-γ agonist of the thiazolidinedione antidiabetics). In the present study, we investigated the effects of rosiglitazone on the development of nonseptic shock caused by zymosan in mice. DESIGN: Exptl. study. SETTING: University laboratory SUBJECTS: Male CD mice. INTERVENTIONS: We investigated the effects of rosiglitazone (3 mg/kg) on the development of nonseptic shock caused by zymosan (500 mg/kg, administered i.p. as a suspension in saline) in mice. MEASUREMENTS AND MAIN RESULTS: Organ failure and systemic inflammation in rats were assessed 18 h after administration of zymosan and/or rosiglitazone and monitored for 12 days (for loss of body weight and mortality rate). Treatment of mice with rosiglitazone (3 mg/kg i.p., 1 and 6 h after zymosan) attenuated the peritoneal exudation and the migration of polymorphonuclear cells caused by zymosan. Rosiglitazone also attenuated the lung, liver, and pancreatic injury and renal dysfunction caused by zymosan as well as the increase in myeloperoxidase activity and malondialdehyde concns. caused by zymosan in the lung and intestine. Immunohistochem. anal. for inducible nitric oxide synthase, nitrotyrosine, and poly(ADP-ribose) revealed pos. staining in lung and intestine tissues obtained from zymosan-treated mice. The degree of staining for nitrotyrosine, inducible nitric oxide synthase, and poly(ADP-ribose) was markedly reduced in tissue sections obtained from zymosan-treated mice that received rosiglitazone. To elucidate whether the protective effects of rosiglitazone are related to activation of the PPAR-γ receptor, we also investigated the effect of a PPAR-γ antagonist, GW 9662, on the protective effects of rosiglitazone. GW 9662 (1 mg/kg administered i.p. 30 mins before treatment with rosiglitazone) significantly antagonized the effect of the PPAR-γ agonist and thus abolished the protective effect. CONCLUSIONS: This study provides evidence, for the first time, that rosiglitazone attenuates the degree of zymosan-induced nonseptic shock in mice.
AN 2004:86506 CAPLUS <<LOGINID::20070323>>
DN 141:199890
TI Rosiglitazone, a ligand of the peroxisome proliferator-activated receptor-γ, reduces the development of nonseptic shock induced by zymosan in mice
AU Cuzzocrea, Salvatore; Pisano, Barbara; Dugo, Laura; Ianaro, Angela; Patel, Nimesh S. A.; Di Paola, Rosanna; Genovese, Tiziana; Chatterjee, Prabal K.; Fulia, Francesco; Cuzzocrea, Elisabetta; Di Rosa, Massimo; Caputi, Achille P.; Thiemermann, Christoph
CS Department of clinical and Experimental Medicine and Pharmacology, Policlinico Universitario, Messina, Italy
SO Critical Care Medicine (2004), 32(2), 457-466
CODEN: CCMDC7; ISSN: 0090-3493
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Thiazolidinediones, a class of anti-diabetic drugs, inhibit Id2 expression through a PPAR.gamma.-independent pathway in human aortic smooth muscle cells

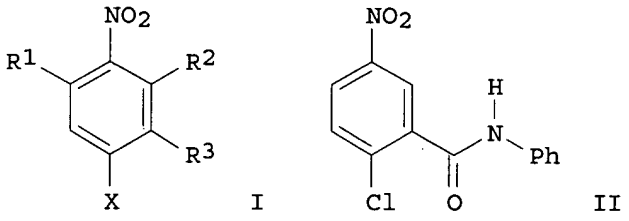
AB Inhibitor of DNA binding (Id2) is a member of the helix-loop-helix family of transcription regulators that is known to play important roles in the proliferation and differentiation of many cell types. Overexpression of Id2 has been reported to result in significant enhancement of vascular smooth muscle cell growth via increased S phase entry. We hypothesized that downregulation of Id2 gene expression by thiazolidinediones (TZDs), a class of anti-diabetic drugs and peroxisome proliferator-activated receptor γ (PPAR.gamma.) activators, might contribute to the anti-atherosclerotic and anti-hypertensive effects of the PPAR γ. Here we document that TZDs, including troglitazone and ciglitazone, repress Id2 gene expression in a doses- and time-dependent manner. However, GW7845, a high-affinity and non-TZD PPAR γ activator, had no inhibitory effect on Id2 gene expression. In addition, PPAR.gamma. antagonist GW9662 did not rescue TZD-induced Id2 repression. Taken together, our data suggest that TZDs repress Id2 expression through a PPAR.gamma.-independent pathway.
AN 2003:199053 CAPLUS <<LOGINID::20070323>>
DN 139:78835
TI Thiazolidinediones, a class of anti-diabetic drugs, inhibit Id2 expression through a PPAR.gamma.-independent pathway in human aortic smooth muscle cells
AU Zhu, X.; Lin, Y.; Zhang, J.; Fu, M.; Mao, Z.; Chen, Y. E.
CS Cardiovascular Research Institute, Peking University Health Science Center, Beijing, 100083, Peop. Rep. China
SO Cellular and Molecular Life Sciences (2003), 60(1), 212-218
CODEN: CMLSFI; ISSN: 1420-682X
PB Birkhaeuser Verlag
DT Journal
LA English
RE.CNT 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Functional Consequences of Cysteine Modification in the Ligand Binding Sites of Peroxisome Proliferator Activated Receptors by GW9662
AB In the course of a high throughput screen to search for ligands of peroxisome proliferator activated receptor-γ (PPAR γ), the authors identified GW9662 using a competition binding assay against the human ligand binding domain. GW9662 had nanomolar IC50 vs. PPAR.gamma. and was 10- and 600-fold less potent in binding expts. using PPAR.alpha. and PPAR.delta., resp. Pretreatment of all three PPARs with GW9662 resulted in the irreversible loss of ligand binding as assessed by scintillation proximity assay. Incubation of PPAR with GW9662 resulted in a change in the absorbance spectra of the receptors consistent with covalent modification. Mass spectrometric anal. of the PPAR.gamma. ligand binding domain treated with GW9662 established Cys285 as the site of covalent modification. This cysteine is conserved among all three PPARs. In cell-based reporter assays, GW9662 was a potent and selective antagonist of full-length PPAR.gamma.. The functional activity of GW9662 as an antagonist of PPAR.gamma. was confirmed in an assay of adipocyte differentiation. GW9662 showed essentially no effect on transcription when tested using both full-length PPAR.delta. and PPAR α. Time-resolved fluorescence assays of ligand-modulated receptor heterodimerization, coactivator binding, and corepressor binding were consistent with the effects observed in the reporter gene assays. Control activators increased PPAR: RXR heterodimer formation and coactivator binding to both PPAR.gamma. and PPAR δ. Corepressor binding was decreased. In the case of PPAR α, GW9662 treatment did not significantly increase heterodimerization and coactivator binding or decrease corepressor binding. The exptl. data indicate that GW9662 modification of each of the three PPARs results in different functional consequences. The selective and irreversible nature of GW9662 treatment, and the observation that

activity is maintained in cell culture expts., suggests that this compound may be a useful tool for elucidation of the role of PPAR.gamma. in biol. processes.

AN 2002:319264 CAPLUS <<LOGINID::20070323>>
DN 137:60888
TI Functional Consequences of Cysteine Modification in the Ligand Binding Sites of Peroxisome Proliferator Activated Receptors by GW9662
AU Leesnitzer, Lisa M.; Parks, Derek J.; Bledsoe, Randy K.; Cobb, Jeff E.; Collins, Jon L.; Consler, Thomas G.; Davis, Roderick G.; Hull-Ryde, Emily A.; Lenhard, James M.; Patel, Lisa; Plunket, Kelli D.; Shenk, Jennifer L.; Stimmel, Julie B.; Therapontos, Christina; Willson, Timothy M.; Blanchard, Steven G.
CS Systems Research Gene Expression and Protein Biochemistry Strategy and Operations CVU CEDD High Throughput Chemistry Proteomics Metabolic Diseases and Molecular Screening, GlaxoSmithKline, Research Triangle Park, NC, 27709, USA
SO Biochemistry (2002), 41(21), 6640-6650
CODEN: BICHAW; ISSN: 0006-2960
PB American Chemical Society
DT Journal
LA English
RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2007 ACS on STN
TI Novel nitroaryl amides as nuclear receptor arylating compounds
GI



AB Nitroaryl amides I [X = halogen; R1 = H, OMe; R2 or R3 = H, other = CONR4R5 (R4 = hydrophobic organic group with MW less than 500 Daltons; R5 = H, alkyl, (un)substituted phenyl)] are disclosed in this invention as novel nuclear receptor ligands. These compds. are useful for arylating a cysteine in a nuclear receptor. Anal. of apparent binding affinities revealed nitroaryl amide II was selective to PPAR.gamma. (resp., binding affinity to PPAR.alpha. and PPAR.delta. was .apprx.10 fold and .apprx.600 fold lower). Binding of II to the cysteine residue in the ligand binding domain of PPAR.gamma. was verified by digestion of the modified PPAR.gamma. with proteolytic enzyme and subsequent sequencing of the resulting peptides using mass spectrometry techniques.

AN 2000:666693 CAPLUS <<LOGINID::20070323>>
DN 133:252168
TI Novel nitroaryl amides as nuclear receptor arylating compounds
IN Blanchard, Steven Gerard; Cobb, Jeffery Edmund; Collins, Jon Loren; Willson, Timothy Mark
PA Glaxo Group Limited, UK
SO PCT Int. Appl., 23 pp.
CODEN: PIXXD2
DT Patent

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LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.          DATE
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PI   WO 2000055118         A1    20000921    WO 2000-US6537           20000313
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
             CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID,
             IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV,
             MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG,
             SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW,
             AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     EP 1161412             A1    20011212    EP 2000-916297           20000313
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             IE, SI, LT, LV, FI, RO
     JP 2002539185          T     20021119    JP 2000-605549           20000313
PRAI US 1999-124635P       P     19990316
     WO 2000-US6537        W     20000313
OS   MARPAT 133:252168
RE.CNT  21    THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s (neointima or atherosclerosis) and (PPAR or
(peroxisome(w)proliferator-activated(w)gamma))
          1779 NEOINTIMA
         53556 ATHEROSCLEROSIS
          8826 PPAR
         17707 PEROXISOME
         11527 PROLIFERATOR
        518979 ACTIVATED
         10513 PROLIFERATOR-ACTIVATED
                 (PROLIFERATOR(W)ACTIVATED)
        843860 GAMMA
            17 PEROXISOME(W)PROLIFERATOR-ACTIVATED(W)GAMMA
L11        953 (NEOINTIMA OR ATHEROSCLEROSIS) AND (PPAR OR (PEROXISOME(W)PROLIF
               ERATOR-ACTIVATED(W)GAMMA))

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       4012080 PY>2003
L12        377 L11 NOT PY>2003

=> s l12 and lysophosphatidic acid
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       4340571 ACID
          2440 LYSOPHOSPHATIDIC ACID
                 (LYSOPHOSPHATIDIC(W)ACID)
L13           0 L12 AND LYSOPHOSPHATIDIC ACID

=> s l12 and (lysophosphatidic acid)
          3115 LYSOPHOSPHATIDIC
       4340571 ACID
          2440 LYSOPHOSPHATIDIC ACID
                 (LYSOPHOSPHATIDIC(W)ACID)
L14           0 L12 AND (LYSOPHOSPHATIDIC ACID)

=> file registry
COST IN U.S. DOLLARS                              SINCE FILE      TOTAL
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FULL ESTIMATED COST                                  82.80     153.11

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                                                        ENTRY    SESSION
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http://www.cas.org/ONLINE/UG/regprops.html

=> exp lysophosphatidic acid/cn
E1            1     LYSOPHOSPHATIDE ACYLTRANSFERASE/CN
E2            1     LYSOPHOSPHATIDES, LYSOCARDIOLIPINS, CATTLE HEART, SODIUM SAL
                    TS/CN
E3            0 --> LYSOPHOSPHATIDIC ACID/CN
E4            1     LYSOPHOSPHATIDIC ACID ACYL TRANSFERASE (HUMAN SEQUENCE HOMOL
                    OG)/CN
E5            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE/CN
E6            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (BDELLOVIBRIO BACTERIO
                    VORUS STRAIN HD100)/CN
E7            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN CLONE MGC:59880
                     IMAGE:6649895)/CN
E8            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN CLONE MGC:71254
                     IMAGE:6577569)/CN
E9            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN ISOENZYME LPAAT
                    -Z)/CN
E10           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (KUENENIA STUTTGARTIEN
                    SIS GENE NLAB)/CN
E11           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (MOUSE STRAIN FVB/N CL
                    ONE MGC:28958 IMAGE:4457846)/CN
E12           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (ORYZA SATIVA JAPONICA
                     GENE OSJNBA0017E08.6)/CN

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                     0.45     153.56

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                     0.00      -8.58

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
       AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
       CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
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       DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 16:54:08 ON 23 MAR 2007

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s (lysophosphatidic(w)acid)
          8   FILE ADISINSIGHT
         97   FILE AGRICOLA
         18   FILE ANABSTR
         12   FILE AQUASCI
         64   FILE BIOENG
       2657   FILE BIOSIS
         86   FILE BIOTECHABS
         86   FILE BIOTECHDS
        790   FILE BIOTECHNO
        106   FILE CABA
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=> s (lysophosphatidic(w)acid) and neointima
          7   FILE BIOSIS
          2   FILE BIOTECHNO
          7   FILE CAPLUS
          1   FILE DDFU
          4   FILE DGENE
  23 FILES SEARCHED...
          1   FILE DISSABS
          1   FILE DRUGU
          4   FILE EMBASE
          3   FILE ESBIOBASE
          1   FILE IFIPAT
          1   FILE LIFESCI
          4   FILE MEDLINE
          1   FILE PASCAL
  53 FILES SEARCHED...
          8   FILE SCISEARCH
          4   FILE TOXCENTER
          8   FILE USPATFULL
          1   FILE WPIDS
          1   FILE WPINDEX

  18 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L15  QUE (LYSOPHOSPHATIDIC(W) ACID) AND NEOINTIMA

=> file biosis
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
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FULL ESTIMATED COST                               1.26     154.82

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                                                 ENTRY    SESSION
CA SUBSCRIBER PRICE                               0.00      -8.58

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CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 21 March 2007 (20070321/ED)
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=> s (lysophosphatidic(w)acid) and neointima
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          2863 NEOINTIMA
L16          7 (LYSOPHOSPHATIDIC(W)ACID) AND NEOINTIMA

=> d l16 1-7 ti

L16  ANSWER 1 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   G protein-coupled receptor kinase 5 inhibits vascular postintervention
     restenosis.

L16  ANSWER 2 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Thrombogenic and atherogenic activities of lysophosphatidic
     acid.

L16  ANSWER 3 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Lysophosphatidic Acid Induces Neointima
     Formation Through PPARg Activation.

L16  ANSWER 4 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Lysophosphatidic acid induces neointima
     formation through PPARgamma activation.

L16  ANSWER 5 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Vascular remodeling induced by naturally occurring unsaturated
     lysophosphatidic acid in vivo.

L16  ANSWER 6 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Lysophosphatidic acid-induced neointima
     formation - the role of PPARgamma.

L16  ANSWER 7 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Athero- and thrombogenic actions of lysophosphatidic
     acid and sphingosine-1-phosphate.


=> d l16 2 3 4 5 6 7 ti abs bib

L16  ANSWER 2 OF 7  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
TI   Thrombogenic and atherogenic activities of lysophosphatidic
     acid.
AB   Lysophosphatidic acid (LPA) has been identified as a
     biologically active lipid in mildly-oxidized LDL, human atherosclerotic
     lesions, and the supernatant of activated platelets.  The evidence that
     LPA has thrombogenic and atherogenic activities has increased
     substantially in recent years.  Supporting the thrombogenic activity of
     LPA, analysis of the core region of human carotid plaques revealed
     recently the presence of alkyl- and acyl-molecular species from LPA with
     high platelet-activating potency (16:0 alkyl-LPA, 20:4 acyl-LPA).  LPA,
     lipid extracts of atherosclerotic plaques, and the lipid-rich core
     elicited shape change and, in synergy with other platelet stimuli,
     aggregation of isolated platelets.  This effect was completely abrogated
     by prior incubation of platelets with LPA receptor antagonists.
     Furthermore, LPA at concentrations approaching those found in vivo,
     induced platelet shape change, aggregation, and platelet-monocyte
     aggregate formation in blood.  LPA-stimulated platelet aggregation was
     mediated by the ADP-stimulated activation of the P2Y1 and P2Y12 receptors.
     Supporting its atherogenic activity, LPA is a mitogen and motogen to
     vascular smooth muscle cells (VSMCs) and an activator of endothelial cells
     and macrophages.  Recently, LPA has been identified as an agonist of the
     peroxisome proliferator activating receptor gamma (PPARgamma), which is a
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key regulator of atherogenesis. LPA elicits progressive neointima formation, which is fully abolished by GW9662, an antagonist of PPARgamma. We propose that LPA plays a central role in eliciting vascular remodeling and atherogenesis. Furthermore, upon rupture of lipid-rich atherosclerotic plaques, LPA may trigger platelet aggregation and intra-arterial thrombus formation. Antagonists of LPA receptors might be useful in preventing LPA-elicited thrombus formation and neointima formation in patients with cardiovascular diseases.

AN 2004:387141 BIOSIS <<LOGINID::20070323>>
DN PREV200400386108
TI Thrombogenic and atherogenic activities of lysophosphatidic acid.
AU Siess, Wolfgang; Tigyi, Gabor [Reprint Author]
CS Ctr Hlth SciDept Physiol, Univ Tennessee, 894 Union Ave, Memphis, TN, 38163, USA
gtigyi@physio1.utmem.edu
SO Journal of Cellular Biochemistry, (August 15 2004) Vol. 92, No. 6, pp. 1086-1094. print.
ISSN: 0730-2312 (ISSN print).
DT Article
LA English
ED Entered STN: 29 Sep 2004
Last Updated on STN: 29 Sep 2004

L16 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Lysophosphatidic Acid Induces Neointima Formation Through PPARg Activation.
AB Neointimal lesions are characterized by accumulation of cells within the arterial wall and are a prelude to atherosclerotic disease that can lead to heart attack and stroke. Here we report that a brief exposure to either alkyl ether analogs of the growth factor-like phospholipid lysophosphatidic acid (LPA), products generated during the oxidative modification of low density lipoprotein, or to unsaturated acyl forms of LPA induce progressive formation of neointima in vivo in a rat carotid artery model. This effect is completely inhibited by pretreatment with the peroxisome proliferator-activated receptor ? (PPAR?) antagonist GW9662 and mimicked by PPAR? agonists Rosiglitazone and 1-O-hexadecyl-2-azaleoyl-phosphatidylcholine. In contrast to these lipids, the PPAR alpha-selective stearoyl-oxovaleryl phosphatidylcholine or EGF, PDGF and VEGF failed to elicit neointima. The structure-activity relationship for neointima induction by LPA analogs in vivo is identical to that of PPAR???activation in vitro and disparate from that of LPA G protein-coupled receptor activation. Neointima-inducing LPA analogs upregulated the CD36 scavenger receptor in vitro and in vivo. These results suggest that select LPAs are important novel endogenous PPAR???ligands capable of mediating vascular remodeling and that activation of the nuclear transcription factor PPAR? is both necessary and sufficient for neointima formation by components of oxidized LDL.
AN 2004:286619 BIOSIS <<LOGINID::20070323>>
DN PREV200400285376
TI Lysophosphatidic Acid Induces Neointima Formation Through PPARg Activation.
AU Tigyi, Gabor J [Reprint Author]; Zhang, Chunxiang; Baker, Daniel; Yasuda, Satoshi; Makarova, Natalia; Balazs, Louisa; Johnson, Leonard; Marathe, Gopal; McIntyre, Thomas; Xu, Yong; Prestwich, Glenn D; Byuin, Hoe-sup; Bittman, Robert
CS Physiology, UTHSC, 894 Union Ave, Memphis, TN, 38163, USA
gtigyi@physio1.utmem.edu
SO FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 276.10.
http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 16 Jun 2004
Last Updated on STN: 16 Jun 2004

L16 ANSWER 4 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Lysophosphatidic acid induces neointima formation through PPARgamma activation.
AB Neointimal lesions are characterized by accumulation of cells within the arterial wall and are a prelude to atherosclerotic disease. Here we report that a brief exposure to either alkyl ether analogs of the growth factor-like phospholipid lysophosphatidic acid (LPA), products generated during the oxidative modification of low density lipoprotein, or to unsaturated acyl forms of LPA induce progressive formation of neointima in vivo in a rat carotid artery model. This effect is completely inhibited by the peroxisome proliferator-activated receptor (PPAR)gamma antagonist GW9662 and mimicked by PPARgamma agonists Rosiglitazone and 1-O-hexadecyl-2-azeleoyl-phosphatidylcholine. In contrast, stearoyl-oxovaleryl phosphatidylcholine, a PPARalpha agonist and polypeptide epidermal growth factor, platelet-derived growth factor, and vascular endothelial growth factor failed to elicit neointima . The structure-activity relationship for neointima induction by LPA analogs in vivo is identical to that of PPARgamma activation in vitro and disparate from that of LPA G protein-coupled receptor activation. Neointima-inducing LPA analogs up-regulated the CD36 scavenger receptor in vitro and in vivo and elicited dedifferentiation of cultured vascular smooth muscle cells that was prevented by GW9662. These results suggest that selected LPA analogs are important novel endogenous PPARgamma ligands capable of mediating vascular remodeling and that activation of the nuclear transcription factor PPARgamma is both necessary and sufficient for neointima formation by components of oxidized low density lipoprotein.
AN 2004:258926 BIOSIS <<LOGINID::20070323>>
DN PREV200400258122
TI Lysophosphatidic acid induces neointima formation through PPARgamma activation.
AU Zhang, Chunxiang; Baker, Daniel L.; Yasuda, Satoshi; Makarova, Natalia; Balazs, Louisa; Johnson, Leonard R.; Marathe, Gopal K.; McIntyre, Thomas M.; Xu, Yong; Prestwich, Glenn D.; Byun, Hoe-Sup; Bittman, Robert; Tigyi, Gabor [Reprint Author]
CS Dept. of Physiology, University of Tennessee Health Science Center, 894 Union Ave., Memphis, TN, 38163, USA
gtigyi@physio1.utmem.edu
SO Journal of Experimental Medicine, (March 15 2004) Vol. 199, No. 6, pp. 763-774. print.
ISSN: 0022-1007 (ISSN print).
DT Article
LA English
ED Entered STN: 19 May 2004
Last Updated on STN: 19 May 2004

L16 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Vascular remodeling induced by naturally occurring unsaturated lysophosphatidic acid in vivo.
AB Background: We previously identified unsaturated (16:1, 18:1, and 18:2) but not saturated (12:0, 14:0, 16:0, and 18:0) lysophosphatidic acids (LPAs) as potent factors for vascular smooth muscle cell (VSMC) dedifferentiation. Unsaturated LPAs strongly induce VSMC dedifferentiation via the coordinated activation of the extracellular signal-regulated-kinase (ERK) and p38 mitogen-activated protein kinase (p38MAPK), resulting in the proliferation and migration of dedifferentiated VSMCs. Here, we investigated the effects of 18:1 and 18:0 LPAs (as representative unsaturated and saturated LPAs, respectively)

on the vasculature in vivo. Methods and Results: Rat common carotid arteries (CCAs) were treated transiently with 18:1 or 18:0 LPA and then examined by histological and biochemical analyses. The 18:1 but not 18:0 LPA potently induced vascular remodeling that was composed primarily of neointima. The incorporation of (3H)18:1 LPA into the CCAs revealed that a sufficient amount of unmetabolized (3H)18:1 LPA to induce VSMC dedifferentiation was present in the vascular wall. The 18:1 LPA-induced neointimal formation in vivo was also dependent on the coordinated activation of ERK and p38MAPK. Unlike balloon-injured CCAs, the 18:1 LPA-treated CCAs showed a histological similarity to human atherosclerotic arteries. Conclusions: This is the first report demonstrating a role for a naturally occurring unsaturated LPA in inducing vascular remodeling in vivo and provides a novel animal model for neointimal formation.

AN 2003:523078 BIOSIS <<LOGINID::20070323>>
DN PREV200300510925
TI Vascular remodeling induced by naturally occurring unsaturated lysophosphatidic acid in vivo.
AU Yoshida, Kenji; Nishida, Wataru; Hayashi, Ken'ichiro; Ohkawa, Yasuyuki; Ogawa, Akira; Aoki, Junken; Arai, Hiroyuki; Sobue, Kenji [Reprint Author]
CS Department of Neuroscience, Graduate School of Medicine, Osaka University, 2-2 Yamadaoka, D13, Suita, Osaka, 565-0871, Japan
sobue@nbiochem.med.osaka-u.ac.jp
SO Circulation, (October 7 2003) Vol. 108, No. 14, pp. 1746-1752. print.
ISSN: 0009-7322 (ISSN print).
DT Article
LA English
ED Entered STN: 5 Nov 2003
Last Updated on STN: 5 Nov 2003

L16 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Lysophosphatidic acid-induced neointima formation - the role of PPARgamma.
AB Lysophosphatidic acid (LPA) induces cellular responses in atherogenesis, including proliferation and dedifferentiation of vascular smooth muscle cells. LPA is generated from activated platelets, after minimal oxidization of LDL, and is enriched in atherosclerotic plaques. LPA activates three G protein-coupled endothelial differentiation gene (EDG) receptors, as well as the intracellular receptor PPARgamma (McIntyre, PNAS 2003).)We examined whether LPA-induced activation of PPARgamma accounts for neointima formation. External carotid arteries of male Sprague-Dawley rats and C57B6 mice were canulated, and the common and internal carotid arteries were clipped. LPA or solvent was infused into the vessel over 30-60 min. Circulation was restored before histological evaluation of the treated vessels occurred 7 to 14 d postsurgery. We found LPAs with unsaturated fatty acids (18:1, 18:2 and 20:4) at 1-10muM caused rapid neointima formation. EDG receptor ligands 18:0 LPA 16:0 LPA, 18:1 cyclic-phosphatidic acid and a hyperactive LPA3 receptor-selective ligand were inactive. In contrast PPARgamma agonists were: fluorinated analogs of LPA (that do not activate EDG receptors at low muM concentrations) and Rosiglitasone (3-10muM) induced a profound neointima. Polypeptide growth factors, PDGF and VEGF (10 ng/ml), and EGF (100 ng/ml), were ineffective in inducing neointima formation.
AN 2003:358665 BIOSIS <<LOGINID::20070323>>
DN PREV200300358665
TI Lysophosphatidic acid-induced neointima formation - the role of PPARgamma.
AU Zhang, Chunxiang [Reprint Author]; Baker, Daniel L.; Balazs, Louisa; Johnson, Leonard R.; McIntyre, Thomas M.; Xu, Yong; Preswitch, Glenn D.; Tigyi, Gabor
CS Medicine, University of Tennessee Health Sciences Center, 894 Union Ave, Memphis, TN, 38163, USA
czhang@utmem.edu; dbaker@physio1.utmem.edu; lbalazs@utmem.edu;

ljohn@physio1.utmem.edu; tom.mcintyre@hmbg.utah.edu;
yong.xu@pharm.utah.edu; gprestwich@pharm.utah.edu;
gtigyi@physio1.utmem.edu
SO FASEB Journal, (March 2003) Vol. 17, No. 4-5, pp. Abstract No. 628.8.
http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. San Diego, CA, USA. April 11-15, 2003. FASEB.
ISSN: 0892-6638 (ISSN print).
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 6 Aug 2003
Last Updated on STN: 6 Aug 2003

L16 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Athero- and thrombogenic actions of lysophosphatidic acid and sphingosine-1-phosphate.
AB Lysophosphatidic acid (LPA) and sphingosine-1-phosphate (S1P) are potent bioactive phospholipids with specific and multiple effects on blood cells and cells of the vessel wall. Released by activated platelets, LPA and S1P mediate physiological wound healing processes such as vascular repair. Evidence is accumulating that these lipid mediators can, however, under certain conditions become athero- and thrombogenic molecules that might aggravate cardiovascular disease. For example, LPA present in minimally modified LDL and within the intima of atherosclerotic lesions may play a role in the early phase of atherosclerosis by inducing barrier dysfunction and increased monocyte adhesion of the endothelium, as well as in the late phase by triggering platelet activation and intra-arterial thrombus formation upon rupture of the atherosclerotic plaque. Moreover, LPA and S1P, by stimulating the proliferation of fibroblasts and by enhancing the survival of inflammatory cells are likely to play a central role in the excessive fibroproliferative and inflammatory response to vascular injury that characterizes the progression of atherosclerosis. Furthermore, LPA can cause the phenotypic dedifferentiation of medial vascular smooth muscle cells, and S1P is able to stimulate the migration and proliferation of intimal vascular smooth muscle cells; both processes ultimately lead to the formation of the neointima. Most importantly, as LPA and S1P bind to and activate multiple G-protein receptors, it emerges that the beneficial or harmful action of LPA and S1P are critically dependent on the expression profile of their receptor subtypes and their coupling to different signal transduction pathways in the target cells. By targeting specific subtypes of LPA and S1P receptors in selective cells of the vascular wall and blood, new strategies for the prevention and therapy of cardiovascular diseases can be envisioned.
AN 2002:446390 BIOSIS <<LOGINID::20070323>>
DN PREV200200446390
TI Athero- and thrombogenic actions of lysophosphatidic acid and sphingosine-1-phosphate.
AU Siess, Wolfgang [Reprint author]
CS Medical Faculty, Institute for Prevention of Cardiovascular Diseases, University of Munich, Pettenkoferstr. 9, D-80336, Munich, Germany
wolfgang.siess@klp.med.uni-muenchen.de
SO Biochimica et Biophysica Acta, (23 May, 2002) Vol. 1582, No. 1-3, pp. 204-215. print.
CODEN: BBACAQ. ISSN: 0006-3002.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 21 Aug 2002
Last Updated on STN: 21 Aug 2002

=> file caplus embase

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COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
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                                                           ENTRY    SESSION
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=> s (lysophosphatidic(w)acid) and neointima
L17         11 (LYSOPHOSPHATIDIC(W) ACID) AND NEOINTIMA

=> dup rem L17
PROCESSING COMPLETED FOR L17
L18          8 DUP REM L17 (3 DUPLICATES REMOVED)

=> d l18 1-8 ti

L18  ANSWER 1 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   High-throughput Screening for LPA3 Antagonist Selectivity

L18  ANSWER 2 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   3-D Database Searching for the Identification of Novel LPA1 Antagonists

L18  ANSWER 3 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Lysophosphatidic acid analogs and inhibition of
     neointima formation

L18  ANSWER 4 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN  DUPLICATE 1
TI   Thrombogenic and atherogenic activities of lysophosphatidic
     acid

L18  ANSWER 5 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN  DUPLICATE 2
TI   Lysophosphatidic acid induces neointima
     formation through PPARγ activation

L18  ANSWER 6 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Lysophospholipid receptors

L18  ANSWER 7 OF 8  EMBASE  COPYRIGHT (c) 2007 Elsevier B.V. All rights
     reserved on STN
TI   Vascular remodeling induced by naturally occurring unsaturated
     lysophosphatidic acid in vivo.

L18  ANSWER 8 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN  DUPLICATE 3
TI   Athero- and thrombogenic actions of lysophosphatidic
     acid and sphingosine-1-phosphate


=> d l18 3 4 5 7 8 ti abs bib

L18  ANSWER 3 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN
TI   Lysophosphatidic acid analogs and inhibition of
     neointima formation
AB   The phospholipid growth factor lysophosphatidic acids (LPAs) containing
     unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing
     hydrocarbon chains with more than 4 carbons were capable of inducing a
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rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPARγ, abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPARγ. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPARγ or antagonists of PPARγ that inhibit PPARγ signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.

AN 2004:857161 CAPLUS <<LOGINID::20070323>>
DN 141:343506
TI Lysophosphatidic acid analogs and inhibition of neointima formation
IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA USA
SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004204383	A1	20041014	US 2004-821739	20040409
	AU 2004229467	A1	20041028	AU 2004-229467	20040409
	CA 2521189	A1	20041028	CA 2004-2521189	20040409
	WO 2004091496	A2	20041028	WO 2004-US11016	20040409
	WO 2004091496	A3	20050324		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1613298	A2	20060111	EP 2004-759365	20040409
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
PRAI	US 2003-462274P	P	20030411		
	WO 2004-US11016	W	20040409		

L18 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
TI Thrombogenic and atherogenic activities of lysophosphatidic acid
AB A review. Lysophosphatidic acid (LPA) has been identified as a biol. active lipid in mildly-oxidized LDL, human atherosclerotic lesions, and the supernatant of activated platelets. The evidence that LPA has thrombogenic and atherogenic activities has increased substantially in recent years. Supporting the thrombogenic activity of LPA, anal. of the core region of human carotid plaques revealed recently the presence of alkyl- and acyl-mol. species from LPA with high platelet-activating potency (16:0 alkyl-LPA, 20:4 acyl-LPA). LPA, lipid exts. of atherosclerotic plaques, and the lipid-rich core elicited shape change and, in synergy with other platelet stimuli, aggregation of isolated platelets. This effect was completely abrogated by prior incubation of platelets with LPA receptor antagonists. Furthermore, LPA at concns. approaching those found in vivo, induced platelet shape change, aggregation, and platelet-monocyte aggregate

formation in blood. LPA-stimulated platelet aggregation was mediated by the ADP-stimulated activation of the P2Y1 and P2Y12 receptors. Supporting its atherogenic activity, LPA is a mitogen and motogen to vascular smooth muscle cells (VSMCs) and an activator of endothelial cells and macrophages. Recently, LPA has been identified as an agonist of the peroxisome proliferator activating receptor γ(PPARγ), which is a key regulator of atherogenesis. LPA elicits progressive neointima formation, which is fully abolished by GW9662, an antagonist of PPARγ. We propose that LPA plays a central role in eliciting vascular remodeling and atherogenesis. Furthermore, upon rupture of lipid-rich atherosclerotic plaques, LPA may trigger platelet aggregation and intra-arterial thrombus formation. Antagonists of LPA receptors might be useful in preventing LPA-elicited thrombus formation and neointima formation in patients with cardiovascular diseases.

AN 2004:654161 CAPLUS <<LOGINID::20070323>>
DN 141:171305
TI Thrombogenic and atherogenic activities of lysophosphatidic acid
AU Siess, Wolfgang; Tigyi, Gabor
CS Institute for Prevention of Cardiovascular Diseases, University of Munich, Germany
SO Journal of Cellular Biochemistry (2004), 92(6), 1086-1094
CODEN: JCEBD5; ISSN: 0730-2312
PB Wiley-Liss, Inc.
DT Journal; General Review
LA English
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AB Neointimal lesions are characterized by accumulation of cells within the arterial wall and are a prelude to atherosclerotic disease. Here the authors report that a brief exposure to either alkyl ether analogs of the growth factor-like phospholipid lysophosphatidic acid (LPA), products generated during the oxidative modification of low d. lipoprotein, or to unsatd. acyl forms of LPA induce progressive formation of neointima in vivo in a rat carotid artery model. This effect is completely inhibited by the peroxisome proliferator-activated receptor (PPAR)γ antagonist GW9662 and mimicked by PPARγ agonists Rosiglitazone and 1-O-hexadecyl-2-azeleoylphosphatidylcholine. In contrast, stearoyloxovalerylphosphatidylcholine, a PPARα agonist and the polypeptides epidermal growth factor, platelet-derived growth factor, and vascular endothelial growth factor failed to elicit neointima . The structure-activity relation for neointima induction by LPA analogs in vivo is identical to that of PPARγ activation in vitro and disparate from that of LPA G protein-coupled receptor activation. Neointima-inducing LPA analogs up-regulated the CD36 scavenger receptor in vitro and in vivo and elicited dedifferentiation of cultured vascular smooth muscle cells that was prevented by GW9662. These results suggest that selected LPA analogs are important novel endogenous PPARγ ligands capable of mediating vascular remodeling and that activation of the nuclear transcription factor PPARγ is both necessary and sufficient for neointima formation by components of oxidized low d. lipoprotein.
AN 2004:242383 CAPLUS <<LOGINID::20070323>>
DN 140:373126
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AU Zhang, Chunxiang; Baker, Daniel L.; Yasuda, Satoshi; Makarova, Natalia; Balazs, Louisa; Johnson, Leonard R.; Marathe, Gopal K.; McIntyre, Thomas M.; Xu, Yong; Prestwich, Glenn D.; Byun, Hoe-Sup; Bittman, Robert; Tigyi,

Gabor
CS Vascular Biology Center of Excellence, The University of Tennessee Health Science Center, Memphis, TN, 38163, USA
SO Journal of Experimental Medicine (2004), 199(6), 763-774
CODEN: JEMEAV; ISSN: 0022-1007
PB Rockefeller University Press
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 7 OF 8 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
TI Vascular remodeling induced by naturally occurring unsaturated lysophosphatidic acid in vivo.
AB Background - We previously identified unsaturated (16:1, 18:1, and 18:2) but not saturated (12:0, 14:0, 16:0, and 18:0) lysophosphatidic acids (LPAs) as potent factors for vascular smooth muscle cell (VSMC) dedifferentiation. Unsaturated LPAs strongly induce VSMC dedifferentiation via the coordinated activation of the extracellular signal-regulated kinase (ERK) and p38 mitogen-activated protein kinase (p38MAPK), resulting in the proliferation and migration of dedifferentiated VSMCs. Here, we investigated the effects of 18:1 and 18:0 LPAs (as representative unsaturated and saturated LPAs, respectively) on the vasculature in vivo. Methods and Results - Rat common carotid arteries (CCAs) were treated transiently with 18:1 or 18:0 LPA and then examined by histological and biochemical analyses. The 18:1 but not 18:0 LPA potently induced vascular remodeling that was composed primarily of neointima. The incorporation of [(3)H]18:1 LPA into the CCAs revealed that a sufficient amount of unmetabolized [(3)H]18:1 LPA to induce VSMC dedifferentiation was present in the vascular wall. The 18:1 LPA-induced neointimal formation in vivo was also dependent on the coordinated activation of ERK and p38MAPK. Unlike balloon-injured CCAs, the 18:1 LPA-treated CCAs showed a histological similarity to human atherosclerotic arteries. Conclusions - This is the first report demonstrating a role for a naturally occurring unsaturated LPA in inducing vascular remodeling in vivo and provides a novel animal model for neointimal formation.
AN 2003410011 EMBASE <<LOGINID::20070323>>
TI Vascular remodeling induced by naturally occurring unsaturated lysophosphatidic acid in vivo.
AU Yoshida K.; Nishida W.; Hayashi K.; Ohkawa Y.; Ogawa A.; Aoki J.; Arai H.; Sobue K.
CS Dr. K. Sobue, Department of Neuroscience, Osaka Univ. Grad. Sch. of Med. (D13), 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan.
sobue@nbiochem.med.osaka-u.ac.jp
SO Circulation, (7 Oct 2003) Vol. 108, No. 14, pp. 1746-1752. .
Refs: 17
ISSN: 0009-7322 CODEN: CIRCAZ
CY United States
DT Journal; Article
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
037 Drug Literature Index
LA English
SL English
ED Entered STN: 30 Oct 2003
Last Updated on STN: 30 Oct 2003

L18 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3
TI Athero- and thrombogenic actions of lysophosphatidic acid and sphingosine-1-phosphate
AB A review. Lysophosphatidic acid (LPA) and sphingosine-1-phosphate (S1P) are potent bioactive phospholipids with specific and multiple effects on blood cells and cells of the vessel wall.

Released by activated platelets, LPA and S1P mediate physiol. wound healing processes such as vascular repair. Evidence is accumulating that these lipid mediators can, however, under certain conditions become athero- and thrombogenic mols. that might aggravate cardiovascular disease. For example, LPA present in minimally modified LDL and within the intima of atherosclerotic lesions may play a role in the early phase of atherosclerosis by inducing barrier dysfunction and increased monocyte adhesion of the endothelium, as well as in the late phase by triggering platelet activation and intra-arterial thrombus formation upon rupture of the atherosclerotic plaque. Moreover, LPA and S1P, by stimulating the proliferation of fibroblasts and by enhancing the survival of inflammatory cells are likely to play a central role in the excessive fibroproliferative and inflammatory response to vascular injury that characterizes the progression of atherosclerosis. Furthermore, LPA can cause the phenotypic dedifferentiation of medial vascular smooth muscle cells, and S1P is able to stimulate the migration and proliferation of intimal vascular smooth muscle cells; both processes ultimately lead to the formation of the neointima. Most importantly, as LPA and S1P bind to and activate multiple G-protein receptors, it emerges that the beneficial or harmful action of LPA and S1P are critically dependent on the expression profile of their receptor subtypes and their coupling to different signal transduction pathways in the target cells. By targeting specific subtypes of LPA and S1P receptors in selective cells of the vascular wall and blood, new strategies for the prevention and therapy of cardiovascular diseases can be envisioned.

AN 2002:459264 CAPLUS <<LOGINID::20070323>>
DN 137:199092
TI Athero- and thrombogenic actions of lysophosphatidic acid and sphingosine-1-phosphate
AU Siess, Wolfgang
CS Medical Faculty, Institute for Prevention of Cardiovascular Diseases, University of Munich, Munich, D-80336, Germany
SO Biochimica et Biophysica Acta, Molecular and Cell Biology of Lipids (2002), 1582(1-3), 204-215
CODEN: BBMLFG; ISSN: 1388-1981
PB Elsevier B.V.
DT Journal; General Review
LA English
RE.CNT 160 THERE ARE 160 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L3              STRUCTURE UPLOADED
L4           50 S L3
L5        19567 S L3 SSS FULL

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L6         5385 S L5/THU
L7            3 S L6 AND NEOINTIMA
L8           70 S L6 AND ATHEROSCLEROSIS
L9           27 S L8 NOT PY>2004
L10           1 S L9 AND LYSOPHOSPHATIDIC
L11           0 S L9 AND (PPAR(W)GAMMA)

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L12        2960 S L5
L13          14 S L12 AND NEOINTIMA
L14           1 S L13 AND LYSOPHOSPHATIDIC
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L16         209 S L15 NOT PY>2004
L17          19 S L16 AND LYSOPHOSPHATIDIC

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L19           1 S L18 FAM FULL

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L21           3 S L20 AND ATHEROSCLEROSIS
L22           1 S L20 AND NEOINTIMA
L23           0 S L20 AND STENT
L24          12 S L20 NOT PY>2003

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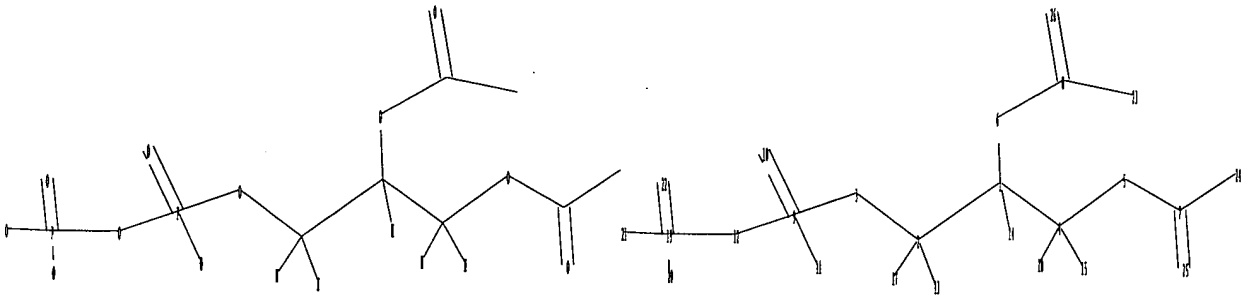

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chain bonds :
1-2  1-4  1-6  1-14  2-3  2-13  2-17  3-9  4-5  4-15  4-18  5-7  6-8  7-24  7-25
8-23  8-26  9-10  9-11  9-12  12-19  19-20  19-21  19-22
exact/norm bonds :
1-6  2-3  3-9  4-5  5-7  6-8  7-25  8-26  9-10  9-11  9-12  12-19  19-20  19-21
19-22
exact bonds :
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G1:C,H,P

Match level :
1:CLASS 2:CLASS 3:CLASS 4:CLASS 5:CLASS 6:CLASS 7:CLASS 8:CLASS 9:CLASS
10:CLASS 11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 17:CLASS 18:CLASS
19:CLASS 20:CLASS
21:CLASS 22:CLASS 23:CLASS 24:CLASS 25:CLASS 26:CLASS

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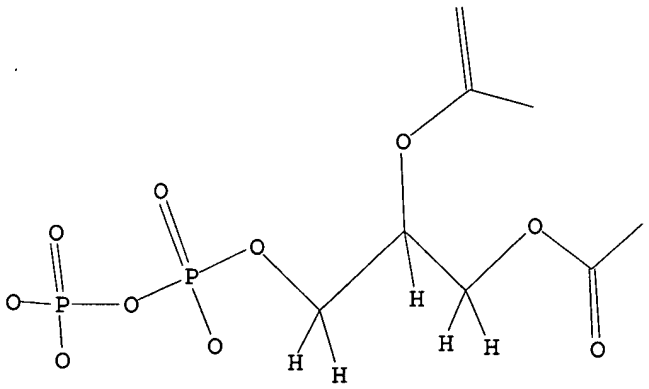
L25 STR



G1 C,H,P

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FULL SUBSET SEARCH INITIATED 17:33:14 FILE 'REGISTRY'
FULL SUBSET SCREEN SEARCH COMPLETED - 184 TO ITERATE

100.0% PROCESSED 184 ITERATIONS 145 ANSWERS
SEARCH TIME: 00.00.01

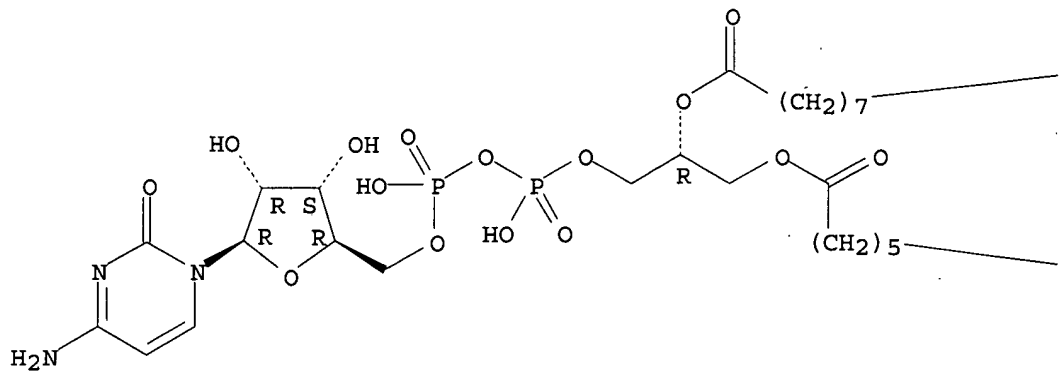
L26 145 SEA SUB=L5 SSS FUL L25

=> d l26 scan

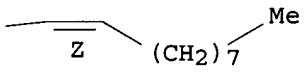
L26 145 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Cytidine 5'-(trihydrogen diphosphate), P'-[(2R)-3-[[6-[(7-nitro-2,1,3-benzoxadiazol-4-yl)amino]-1-oxohexyl]oxy]-2-[[(9Z)-1-oxo-9-octadecenyl]oxy]propyl] ester (9CI)
MF C42 H65 N7 O18 P2

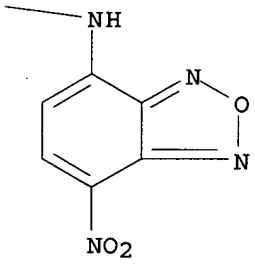
Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



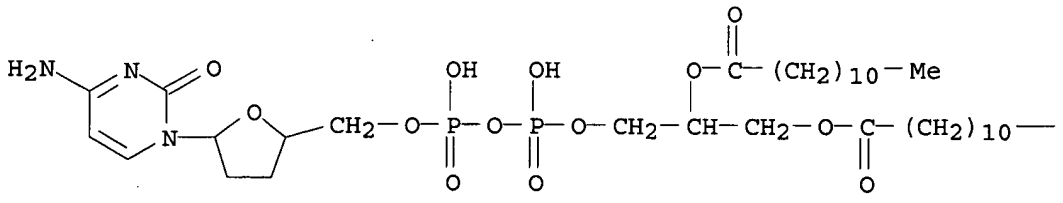


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):3

L26 145 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Cytidine 5'-(trihydrogen diphosphate), 2',3'-dideoxy-, P'-[2,3-bis[(1-oxododecyl)oxy]propyl] ester (9CI)
MF C36 H65 N3 O13 P2

PAGE 1-A



PAGE 1-B

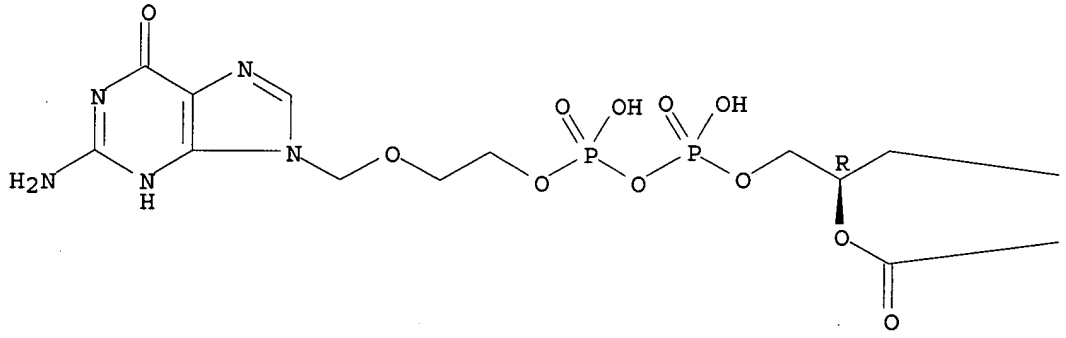
—Me

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

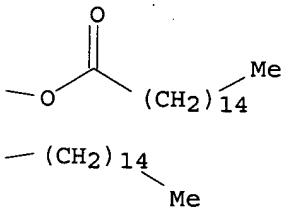
L26 145 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Hexadecanoic acid, 1-[10-(2-amino-1,6-dihydro-6-oxo-9H-purin-9-yl)-3,5-dihydroxy-3,5-dioxido-2,4,6,9-tetraoxa-3,5-diphosphadec-1-yl]-1,2-ethanediyl ester, (R)- (9CI)
MF C43 H79 N5 O13 P2

Absolute stereochemistry.

PAGE 1-A

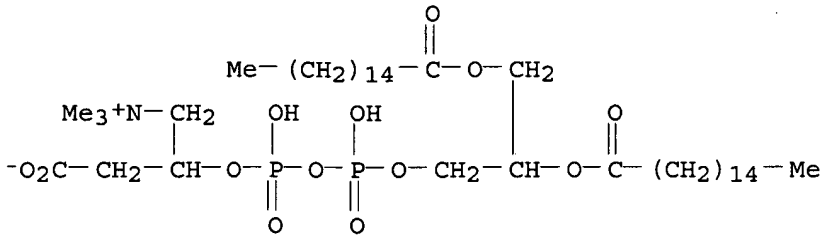


PAGE 1-B



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L26 145 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN 3,5,7,11-Tetraoxa-4,6-diphosphaheptacosan-1-aminium, 2-(carboxymethyl)-4,6-dihydroxy-N,N,N-trimethyl-12-oxo-9-[(1-oxohexadecyl)oxy]-, inner salt, 4,6-dioxide (9CI)
MF C42 H83 N O13 P2



HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	40.28	808.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-15.00

FILE 'CAPLUS' ENTERED AT 17:33:40 ON 11 DEC 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 11 Dec 2006 VOL 145 ISS 25
FILE LAST UPDATED: 10 Dec 2006 (20061210/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l26
L27 111 L26

=> s l27 and (restenosis or neointima)
7958 RESTENOSIS
1737 NEOINTIMA
L28 0 L27 AND (RESTENOSIS OR NEOINTIMA)

=> s l27 and (PPAR(w)gamma)
8370 PPAR
832731 GAMMA
5508 PPAR(W)GAMMA
L29 0 L27 AND (PPAR(W)GAMMA)

=> file registry
COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION
FULL ESTIMATED COST | 8.72 | 817.00

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE ENTRY | TOTAL SESSION
CA SUBSCRIBER PRICE | 0.00 | -15.00

FILE 'REGISTRY' ENTERED AT 17:34:35 ON 11 DEC 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 10 DEC 2006 HIGHEST RN 915124-84-4
DICTIONARY FILE UPDATES: 10 DEC 2006 HIGHEST RN 915124-84-4

New CAS Information Use Policies, enter HELP USAGETERMS for details.

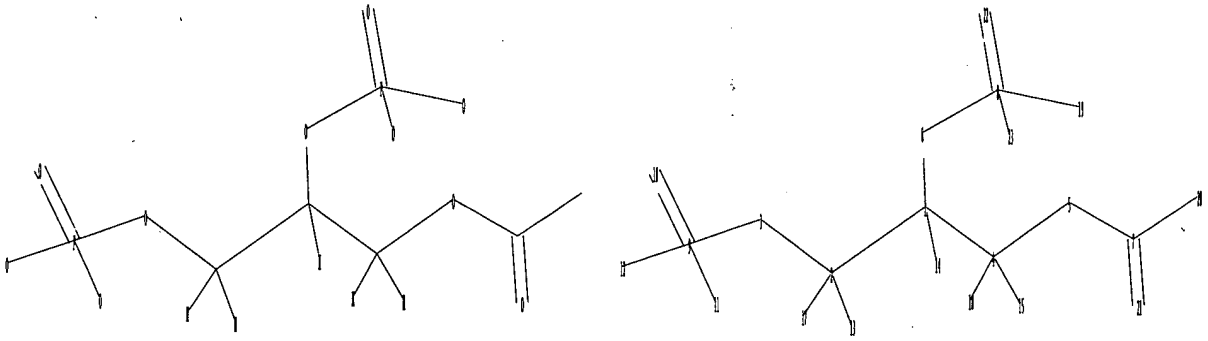
TSCA INFORMATION NOW CURRENT THROUGH June 30, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=>
Uploading C:\Program Files\Stnexp\Queries\10821739MAGDP.str



chain nodes :
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 17 18 19 20 21 22 23
chain bonds :
1-2 1-4 1-6 1-14 2-3 2-13 2-17 3-9 4-5 4-15 4-18 5-7 6-8 7-20 7-21 8-19 8-22 8-23 9-10 9-11 9-12
exact/norm bonds :
1-6 2-3 3-9 4-5 5-7 6-8 7-21 8-19 8-22 8-23 9-10 9-11 9-12
exact bonds :
1-2 1-4 1-14 2-13 2-17 4-15 4-18 7-20

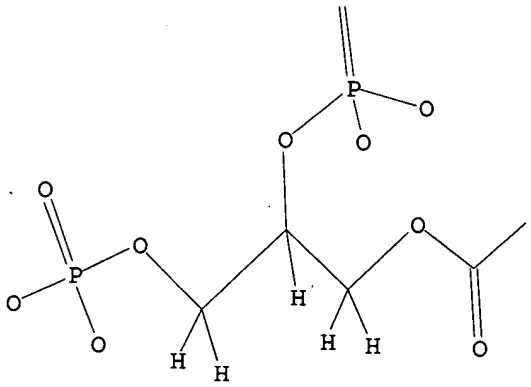
G1:C,H,P

Match level :

1:CLASS 2:CLASS 3:CLASS 4:CLASS 5:CLASS 6:CLASS 7:CLASS 8:CLASS 9:CLASS
10:CLASS 11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 17:CLASS 18:CLASS
19:CLASS 20:CLASS
21:CLASS 22:CLASS 23:CLASS

L30 STRUCTURE UPLOADED

=> d l30
L30 HAS NO ANSWERS
L30 STR



G1 C,H,P

Structure attributes must be viewed using STN Express query preparation.

=> s l30 sub=l5
ENTER SUBSET SEARCH SCOPE - SAMPLE, FULL, RANGE, OR (END):full
FULL SUBSET SEARCH INITIATED 17:36:01 FILE 'REGISTRY'
FULL SUBSET SCREEN SEARCH COMPLETED - 1383 TO ITERATE

100.0% PROCESSED 1383 ITERATIONS 26 ANSWERS
SEARCH TIME: 00.00.01

L31 26 SEA SUB=L5 SSS FUL L30

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	40.28	857.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-15.00

FILE 'CAPLUS' ENTERED AT 17:36:04 ON 11 DEC 2006
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FILE COVERS 1907 - 11 Dec 2006 VOL 145 ISS 25
FILE LAST UPDATED: 10 Dec 2006 (20061210/ED)

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They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l30
REG1stRY INITIATED
Substance data SEARCH and crossover from CAS REGISTRY in progress...
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.

SAMPLE SEARCH INITIATED 17:36:08 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 116 TO ITERATE

100.0% PROCESSED 116 ITERATIONS 1 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 1674 TO 2966
PROJECTED ANSWERS: 1 TO 80

L32 1 SEA SSS SAM L30

L33 3 L32

=> s l31
L34 10 L31

=> d l34 1-10 ti

L34 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid (LPA) receptor agonists and antagonists, their preparation, and methods of use

L34 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid (LPA) receptor agonists and antagonists, their preparation, and methods of use

L34 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Synthesis of lysophosphatidic acid receptor agonists and antagonists and their use for cancer inhibition, wound healing, and enhancement of cell proliferation

L34 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Compositions for sustained release of a antimicrobial gas

L34 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method of making an amine containing biocidal composition

L34 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Synthesis and properties of an organophosphorus regulator of hemoglobin oxygenation

L34 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dental adhesive compositions containing acrylate phosphates

L34 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dental adhesives containing phosphonic acids

L34 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Adhesives for prosthetics

L34 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2006 ACS on STN
TI Adhesive composition

=> s l34 not py>2004
2477817 PY>2004
L35 7 L34 NOT PY>2004

=> d l35 1-7 ti

L35 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Synthesis of lysophosphatidic acid receptor agonists and antagonists and their use for cancer inhibition, wound healing, and enhancement of cell proliferation

L35 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method of making an amine containing biocidal composition

L35 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Synthesis and properties of an organophosphorus regulator of hemoglobin oxygenation

L35 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dental adhesive compositions containing acrylate phosphates

L35 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dental adhesives containing phosphonic acids

L35 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Adhesives for prosthetics

L35 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Adhesive composition

=> d l35 1-7 ti abs bib

L35 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Synthesis of lysophosphatidic acid receptor agonists and antagonists and their use for cancer inhibition, wound healing, and enhancement of cell proliferation
AB The present invention relates to lysophosphatidic acid (LPA) analogs and cyclic derivs. of the analogs as well as pharmaceutical compns. which include those compds. Also disclosed are methods of using such compds., which have activity as agonists or as antagonists of LPA receptors; such methods including inhibiting LPA activity on an LPA receptor, modulating LPA receptor activity, treating cancer, enhancing cell proliferation, and treating a wound. Thus, 2-amino-3-oxo-3-(tetradecylamino)propyl dihydrogen phosphate (I), 2-(acetylamino)-3-oxo-3-(tetradecylamino)propyl dihydrogen phosphate (II), and 1,2-(3-octadecyloxypropane)-bis(dihydrogen

phosphate) (III) were synthesized. The cytotoxicity of these compds. on prostate cancer cell lines was determined The IC50's observed were 0.7 ± 0.1 for I on PC-3 cells, 0.7 ± 0.1 for II on DU145 cells, and 3.1 ± 3.2 for III on LNCaP cells. Addnl., phosphoric acid monododecyl ester (IV) was prepared and screened in Xenopus oocytes (which produce the PSP24 receptor) and in recombinant RH7777 cells producing Edg-2, Edg-4, and Edg-7 receptors. In Xenopus IV inhibited LPA-induced chloride currents with an IC50 value of about 8.1 nM. In Edg-2 and Edg-4-expressing RH7777 cells IV significantly inhibited the Ca2+ responses while in Edg-7-expressing cells this compound increased the Ca2+ responses.

AN 2001:713600 CAPLUS <<LOGINID::20061211>>
DN 135:267219
TI Synthesis of lysophosphatidic acid receptor agonists and antagonists and their use for cancer inhibition, wound healing, and enhancement of cell proliferation
IN Miller, Duane D.; Tigyi, Gabor; Dalton, James T.; Sardar, Vineet M.; Elrod, Don B.; Xu, Huiping; Baker, Daniel L.; Wang, Dean; Liliom, Karoly; Fischer, David J.; Virag, Tamas; Nusser, Nora
PA University of Tennessee Research Corporation, USA
SO PCT Int. Appl., 140 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001071022	A2	20010927	WO 2001-US8729	20010319
	WO 2001071022	A3	20020404		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2402038	AA	20010927	CA 2001-2402038	20010319
	AU 2001049263	A5	20011003	AU 2001-49263	20010319
	EP 1263752	A2	20021211	EP 2001-922465	20010319
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004506604	T2	20040304	JP 2001-569403	20010319
PRAI	US 2000-190370P	P	20000317		
	WO 2001-US8729	W	20010319		

OS MARPAT 135:267219

L35 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method of making an amine containing biocidal composition
AB A process for preparing a composite including mixing a hydrophilic material containing an amine with a hydrophobic material containing an acid releasing agent, the hydrophilic and hydrophobic materials being substantially free of water, to form a mixture The mixture is exposed to chlorine dioxide which reacts with the amine to form iminium chlorite within the hydrophilic material. The hydrophilic material is capable of releasing chlorine dioxide upon hydrolysis of the acid releasing agent.
AN 1997:632706 CAPLUS <<LOGINID::20061211>>
DN 127:283427
TI Method of making an amine containing biocidal composition
IN Wellinghoff, Stephen T.
PA Southwest Research Institute, USA
SO U.S., 29 pp., Cont.-in-part of U.S. Ser. No. 192,499, abandoned.
CODEN: USXXAM
DT Patent

LA English
FAN.CNT 12

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5668185	A	19970916	US 1995-461716	19950605
	US 5360609	A	19941101	US 1993-17657	19930212
	CA 2196782	AA	19961212	CA 1996-2196782	19960604
	WO 9639029	A1	19961212	WO 1996-US9475	19960604
	W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN				
	AU 9661602	A1	19961224	AU 1996-61602	19960604
	AU 698032	B2	19981022		
	EP 774898	A1	19970528	EP 1996-919204	19960604
	EP 774898	B1	20030305		
	R: BE, DE, DK, ES, GB, IE, IT, SE				
	BR 9606416	A	19971014	BR 1996-6416	19960604
	JP 10504844	T2	19980512	JP 1997-501786	19960604
	ES 2193244	T3	20031101	ES 1996-919204	19960604
	US 6046243	A	20000404	US 1997-858860	19970519
	HK 1013215	A1	20030926	HK 1998-114509	19981221
PRAI	US 1993-16904	B3	19930212		
	US 1993-17657	A3	19930212		
	US 1994-192498	B2	19940203		
	US 1994-192499	B2	19940203		
	US 1994-228671	B2	19940418		
	US 1995-461304	A2	19950605		
	US 1995-461706	A	19950605		
	US 1995-461716	A	19950605		
	US 1995-462039	B2	19950605		
	US 1995-462164	A	19950605		
	US 1995-465086	A3	19950605		
	US 1995-465087	B1	19950605		
	US 1995-465358	A2	19950605		
	WO 1996-US9475	W	19960604		
	US 1996-682318	A2	19960717		
	US 1996-724907	A2	19961003		
	US 1996-726413	A2	19961003		

L35 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Synthesis and properties of an organophosphorus regulator of hemoglobin oxygenation
AB Synthesis of D,L-1-O-(ε-aminohexanoyl)-2,3-diphosphoglycerol (ADPG) and characterization of some of its chemical and biol. properties were performed. The pH-dependence of the 31P NMR of tricyclohexylammonium ADPG was examined; the chemical shift of the P in position 2 decreased on acidification. The ADPG salt was not incorporated into phosphatidylcholine (PC) bilayer membranes, but was efficiently trapped within PC liposomes. Titration of ADPG revealed 2 groups with pKa values of 2.8, and 1 with pKa .apprx.6.5. Finally, the interaction of ADPG with Hb was investigated by 31P NMR. The NMR signal of P3 was shifted on association with oxyHb. The O affinity of Hb was estimated as p50O2 (the pO2 for 50% saturation of Hb). In the absence of ADPG, p50O2 was 2.6 kP; addition of ADPG increased p50O2 to 3.5 kP, whereas addition of 2,3-DPG increased p50O2 to 4.4 kP.
AN 1985:162441 CAPLUS <<LOGINID::20061211>>
DN 102:162441
TI Synthesis and properties of an organophosphorus regulator of hemoglobin oxygenation
AU Ushakova, I. P.; Tuvin, M. Yu.; Serebrennikova, G. A.; Kol'tsova, G. N.; Vyazova, E. P.; Rozenberg, G. Ya.; Evstigneeva, R. P.

CS M. V. Lomonosov Inst. Fine Chem. Technol., Moscow, USSR
SO Bioorganicheskaya Khimiya (1985), 11(2), 187-91
CODEN: BIKHD7; ISSN: 0132-3423
DT Journal
LA Russian

L35 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dental adhesive compositions containing acrylate phosphates
AB Dental and surgical adhesives are prepared from phosphoalkyl acryloyl- or acrylamidoalkylcarboxylates and vinyl monomers. CH2:CMeCOCl [920-46-7] and 12-aminododecanoic acid [693-57-2] were treated to give N-methacryloyl-12-aminododecanoic acid [62839-65-0] which was mixed with glycidol [556-52-5] and MeCH2N+Et3Cl- to give CH2:CMeCONH(CH2)11CO2CH2CH(OH)CH2OH [92915-67-8]. This was treated with POCl3 to give CH2:CMeCONH(CH2)11CO2CH2CH(OPO3H2)CH2OPO3H2 (I) [92900-11-3]. A 2-pad primer was prepared from A: Bis-GMA 50, hydroxyethyl methacrylate 43, I 7, and Bz2O2 2 parts by weight and B: EtOH 100, Na benzenesulfonate 3, and N,N-dimethyl-p-toluidine 0.7 parts by weight The adhesiveness of a mixture of equal amts. of A and B on dentin surface for bonding a dental resin was demonstrated and was superior to a similar composition containing 2-methacryloyloxyethyl dihydrogen phosphate instead of I.
AN 1984:598235 CAPLUS <<LOGINID::20061211>>
DN 101:198235
TI Dental adhesive compositions containing acrylate phosphates
IN Omura, Ikuo; Yamauchi, Junichi; Nagase, Yoshinori; Uemura, Fumiko
PA Kuraray Co., Ltd. , Japan
SO Eur. Pat. Appl., 44 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

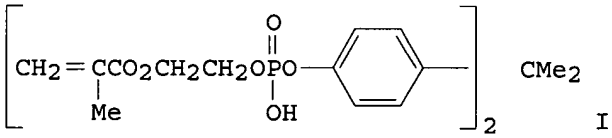
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 115410	A2	19840808	EP 1984-300358	19840120
	EP 115410	A3	19840829		
	EP 115410	B1	19870520		
	R: DE, FR, GB, IT, NL				
	JP 59135272	A2	19840803	JP 1983-9170	19830121
	JP 02029104	B4	19900627		
	US 4499251	A	19850212	US 1984-570292	19840113
	US 4537940	A	19850827	US 1984-659455	19841010
PRAI	JP 1983-9170	A	19830121		
	US 1984-570292	A1	19840113		

L35 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Dental adhesives containing phosphonic acids
AB Polymers for dental adhesives were prepared from polymers containing phosphonic acids. Thus, a powder mixture of 100 weight% poly(Me methacrylate), 2 weight% Bz2O2, and 3 weight% Na p-toluenesulfonate was reacted with a liquid mixture of 80 weight parts Me methacrylate, 10 weight parts ethylene glycol dimethacrylate, 10 weight parts 2-methacryloxyethyl phenylphosphonate, and 1.0 weight part N,N'-diethanol p-toluidine to give a polymer [64716-64-9] which had a strength of 160 kg/cm2 as opposed to 35 kg/cm2 for similar polymers without the phosphonic acid monomers. Poly(bisphenol A diglycidyl methacrylate-2-methacryloxyethyl phenylphosphonate-triethylene glycol dimethacrylate) [64716-49-0], poly(bisphenol A diglycidyl methacrylate-triethylene glycol dimethacrylate) [26426-05-1] and 5 more similar resins were also prepared
AN 1980:625653 CAPLUS <<LOGINID::20061211>>
DN 93:225653
TI Dental adhesives containing phosphonic acids
IN Yamanouchi, Junichi; Masuhara, Eiichi; Nakabayashi, Nobuo; Shibatani,

Koichiro; Wada, Itaru
PA Kuraray Co., Ltd., Japan
SO Jpn. Tokkyo Koho, 15 pp.
CODEN: JAXXAD
DT Patent
LA Japanese
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 55005790	B4	19800209	JP 1976-143807	19761129
	JP 53067740	A2	19780616		
	GB 1569021	A	19800611	GB 1977-10480	19770311
	DE 2711234	A1	19770929	DE 1977-2711234	19770315
	DE 2711234	B2	19810507		
	DE 2711234	C3	19880707		
	FR 2344281	A1	19771014	FR 1977-8015	19770317
	FR 2344281	B1	19810116		
	US 4259117	A	19810331	US 1978-936759	19780825
	US 4259075	A	19810331	US 1979-78048	19790924
	US 4368043	A	19830111	US 1980-149770	19800514
PRAI	JP 1976-30045	A	19760317		
	JP 1976-143807	A	19761129		
	JP 1976-145049	A	19761130		
	US 1977-778734	A1	19770317		
	US 1978-936759	A3	19780825		

L35 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Adhesives for prosthetics
GI



I

AB Adhesives for human hard tissues (such as bones, teeth, etc.) are formulated with phosphoric acid esters (methacryloyloxyalkyl and(or) bisphenol, alkylene, phenylene, etc. esters). E.g., a adhesive consists of (A) diethylene glycol dimethacrylate 20, Bisphenol-A diglycidyl dimethacrylate 30, silane-treated quartz (10-50 μ diameter) 165, I [64716-42-3] 10 and Bz2O2 2 parts; and (B) diethylene glycol dimethacrylate 20, Bisphenol-A diglycidyl dimethacrylate 30, silane-treated quartz (10-50 μ diam) 165, N,N-dimethyl-p-toluidine 2 and p-toluenesulfonic acid Na salt 2 parts. An equal part of A and B had a adhesive strength of 178 kg/cm2 after mixing.
AN 1978:552750 CAPLUS <<LOGINID::20061211>>
DN 89:152750
TI Adhesives for prosthetics
IN Yamanouchi, Junichi; Masuhara, Eiichi; Nakabayashi, Nobuo; Shibatani, Koichiro; Wada, Itaru
PA Kuraray Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 15 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 53069494	A2	19780620	JP 1976-145049	19761130

	JP 55002235	B4	19800118		
	GB 1569021	A	19800611	GB 1977-10480	19770311
	DE 2711234	A1	19770929	DE 1977-2711234	19770315
	DE 2711234	B2	19810507		
	DE 2711234	C3	19880707		
	FR 2344281	A1	19771014	FR 1977-8015	19770317
	FR 2344281	B1	19810116		
	US 4259117	A	19810331	US 1978-936759	19780825
	US 4259075	A	19810331	US 1979-78048	19790924
	US 4368043	A	19830111	US 1980-149770	19800514
PRAI	JP 1976-30045	A	19760317		
	JP 1976-143807	A	19761129		
	JP 1976-145049	A	19761130		
	US 1977-778734	A1	19770317		
	US 1978-936759	A3	19780825		

L35 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Adhesive composition
AB Compds. such as RP(O)(OH)OCH2CH2O2CCMe:CH2 (I) (R = OPh or Ph), (H2C:CMeCO2CH2)2CHOP(O)(OH)OPh, and [H2C:CMeCO2CH2CH2OP(O)(OH)OR]2CMe2 (R = p-C6H4) were used in adhesives, filling materials, etc., having good adhesion to dental enamel, bone, etc. Thus, 98 parts powdered poly(Me methacrylate) containing 2 parts Bz2O2 was mixed with 95 parts Me methacrylate containing 5 parts I (R = OPh) and 2.5 parts p-toluenesulfinic acid, applied to ivory, and hardened at 37° for 72 h to give an adhesive [64716-58-1] with bond strength 160 kg/cm2.
AN 1978:7867 CAPLUS <<LOGINID::20061211>>
DN 88:7867
TI Adhesive composition
IN Yamauchi, Junichi; Masuhara, Eiichi; Nakabayashi, Nobuo; Shibatani, Kyoichiro; Wada, Toru
PA Kuraray Co., Ltd., Japan
SO Ger. Offen., 58 pp.
CODEN: GWXXBX
DT Patent
LA German
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 2711234	A1	19770929	DE 1977-2711234	19770315
	DE 2711234	B2	19810507		
	DE 2711234	C3	19880707		
	JP 52113089	A2	19770921	JP 1976-30045	19760317
	JP 55030768	B4	19800813		
	JP 55005790	B4	19800209	JP 1976-143807	19761129
	JP 53067740	A2	19780616		
	JP 53069494	A2	19780620	JP 1976-145049	19761130
	JP 55002235	B4	19800118		
PRAI	JP 1976-30045	A	19760317		
	JP 1976-143807	A	19761129		
	JP 1976-145049	A	19761130		

=> file uspatfull

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	27.66	885.84

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.25	-20.25

FILE 'USPATFULL' ENTERED AT 17:37:31 ON 11 DEC 2006
CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 7 Dec 2006 (20061207/PD)
FILE LAST UPDATED: 7 Dec 2006 (20061207/ED)
HIGHEST GRANTED PATENT NUMBER: US7146645
HIGHEST APPLICATION PUBLICATION NUMBER: US2006277640
CA INDEXING IS CURRENT THROUGH 7 Dec 2006 (20061207/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 7 Dec 2006 (20061207/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Jun 2006
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Jun 2006

=> s l31
L36 10 L31

=> d l36 1-10 ti

L36 ANSWER 1 OF 10 USPATFULL on STN
TI LPA receptor agonists and antagonists and methods of use

L36 ANSWER 2 OF 10 USPATFULL on STN
TI LPA receptor agonists and antagonists and methods of use

L36 ANSWER 3 OF 10 USPATFULL on STN
TI LPA receptor agonists and antagonists and methods of use

L36 ANSWER 4 OF 10 USPATFULL on STN
TI Compositions for sustained release of a gas

L36 ANSWER 5 OF 10 USPATFULL on STN
TI Method of making an amine containing biocidal composition

L36 ANSWER 6 OF 10 USPATFULL on STN
TI Adhesive compositions

L36 ANSWER 7 OF 10 USPATFULL on STN
TI Adhesive compositions

L36 ANSWER 8 OF 10 USPATFULL on STN
TI Adhesive cementing agents for the hard tissues of the human body

L36 ANSWER 9 OF 10 USPATFULL on STN
TI Dental filling material

L36 ANSWER 10 OF 10 USPATFULL on STN
TI Method of filling a tooth cavity

=> d l36 1-3 ti abs bib

L36 ANSWER 1 OF 10 USPATFULL on STN
TI LPA receptor agonists and antagonists and methods of use
AB The present invention relates to compounds according to formula (I) as disclosed herein as well as pharmaceutical compositions which include those compounds. Also disclosed are methods of using such compounds, which have activity as agonists or as antagonists of LPA receptors; such methods including inhibiting LPA activity on an LPA receptor, modulating LPA receptor activity, treating cancer, enhancing cell proliferation, and treating a wound.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AN 2005:299583 USPATFULL <<LOGINID::20061211>>
TI LPA receptor agonists and antagonists and methods of use
IN Miller, Duane D., Germantown, TN, UNITED STATES
Tigyi, Gabor, Memphis, TN, UNITED STATES

Dalton, James T., Columbus, OH, UNITED STATES
Sardar, Vineet M., Cordova, TN, UNITED STATES
Elrod, Don B., College Station, TX, UNITED STATES
Xu, Huiping, Memphis, TN, UNITED STATES
Baker, Daniel L., Memphis, TN, UNITED STATES
Wang, Dean, Memphis, TN, UNITED STATES
Liliom, Karoly, Budapest, HUNGARY
Fischer, David J., Plymouth, MA, UNITED STATES
Virag, Tamas, Memphis, TN, UNITED STATES
Nusser, Nora, Memphis, TN, UNITED STATES
PI US 2005261252 A1 20051124
AI US 2005-67884 A1 20050228 (11)
RLI Division of Ser. No. US 2001-811838, filed on 19 Mar 2001, GRANTED, Pat. No. US 6875757
PRAI US 2000-190370P 20000317 (60)
DT Utility
FS APPLICATION
LREP Edwin V. Merkel, Nixon Peabody LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603-1051, US
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN 26 Drawing Page(s)
LN.CNT 4436
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L36 ANSWER 2 OF 10 USPATFULL on STN
TI LPA receptor agonists and antagonists and methods of use
AB The present invention relates to compounds according to formula (I) as disclosed herein as well as pharmaceutical compositions which include those compounds. Also disclosed are methods of using such compounds, which have activity as agonists or as antagonists of LPA receptors; such methods including inhibiting LPA activity on an LPA receptor, modulating LPA receptor activity, treating cancer, enhancing cell proliferation, treating a wound, treating apoptosis or preserving or restoring function in a cell, tissue, or organ, culturing cells, preserving organ or tissue function, and treating a dermatological condition.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AN 2003:188444 USPATFULL <<LOGINID::20061211>>
TI LPA receptor agonists and antagonists and methods of use
IN Miller, Duane D., Germantown, TN, UNITED STATES
Tigyi, Gabor, Memphis, TN, UNITED STATES
Dalton, James T., Columbus, OH, UNITED STATES
Sardar, Vineet M., Cordova, TN, UNITED STATES
Elrod, Don B., College Station, TX, UNITED STATES
Xu, Huiping, Memphis, TN, UNITED STATES
Baker, Daniel L., Memphis, TN, UNITED STATES
Wang, Dean, Memphis, TN, UNITED STATES
Liliom, Karoly, Budapest, HUNGARY
Fischer, David J., Cordova, TN, UNITED STATES
Virag, Tamas, Memphis, TN, UNITED STATES
Nusser, Nora, Memphis, TN, UNITED STATES
PI US 2003130237 A1 20030710
AI US 2001-953686 A1 20010918 (9)
RLI Continuation-in-part of Ser. No. US 2001-811838, filed on 19 Mar 2001, PENDING
PRAI US 2000-190370P 20000317 (60)
DT Utility
FS APPLICATION
LREP Michael L. Goldman, NIXON PEABODY LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 26 Drawing Page(s)

LN.CNT 4417
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L36 ANSWER 3 OF 10 USPATFULL on STN
TI LPA receptor agonists and antagonists and methods of use
AB The present invention relates to compounds according to formula (I) as disclosed herein as well as pharmaceutical compositions which include those compounds. Also disclosed are methods of using such compounds, which have activity as agonists or as antagonists of LPA receptors; such methods including inhibiting LPA activity on an LPA receptor, modulating LPA receptor activity, treating cancer, enhancing cell proliferation, and treating a wound.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AN 2003:38153 USPATFULL <<LOGINID::20061211>>
TI LPA receptor agonists and antagonists and methods of use
IN Miller, Duane D., Germantown, TN, UNITED STATES
Tigyi, Gabor, Memphis, TN, UNITED STATES
Dalton, James T., Columbus, OH, UNITED STATES
Sardar, Vineet M., Cordova, TN, UNITED STATES
Elrod, Don B., College Station, TX, UNITED STATES
Xu, Huiping, Columbus, OH, UNITED STATES
Baker, Daniel L., Memphis, TN, UNITED STATES
Wang, Dean, Memphis, TN, UNITED STATES
Liliom, Karoly, Budapest, HUNGARY
Fischer, David J., Plymouth, MA, UNITED STATES
Virag, Tamas, Memphis, TN, UNITED STATES
Nusser, Nora, Memphis, TN, UNITED STATES
PI US 2003027800 A1 20030206
US 6875757 B2 20050405
AI US 2001-811838 A1 20010319 (9)
PRAI US 2000-190370P 20000317 (60)
DT Utility
FS APPLICATION
LREP Michael L. Goldman, NIXON PEABODY LLP, Clinton Square, P.O. Box 31051, Rochester, NY, 14603
CLMN Number of Claims: 34
ECL Exemplary Claim: 1
DRWN 26 Drawing Page(s)
LN.CNT 4588
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	10.81	896.65

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-20.25

FILE 'REGISTRY' ENTERED AT 17:39:09 ON 11 DEC 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 10 DEC 2006 HIGHEST RN 915124-84-4
DICTIONARY FILE UPDATES: 10 DEC 2006 HIGHEST RN 915124-84-4

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH June 30, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

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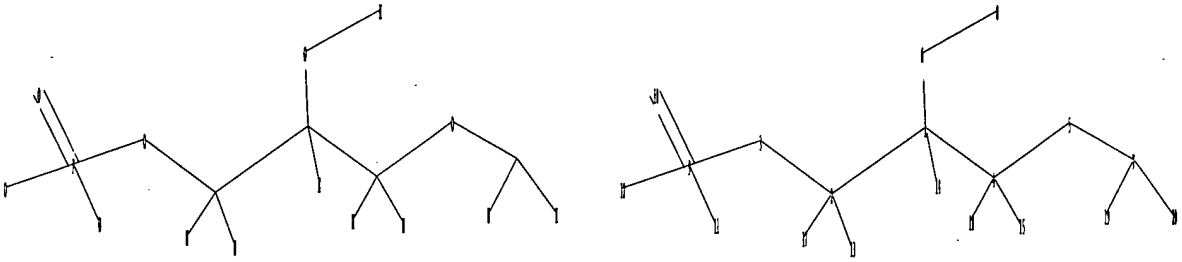

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chain bonds :
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exact/norm bonds :
1-6 2-3 3-9 4-5 5-7 9-10 9-11 9-12
exact bonds :
1-2 1-4 1-14 2-13 2-17 4-15 4-18 6-8 7-20 7-19

G1:C,H,P

Match level :
1:CLASS 2:CLASS 3:CLASS 4:CLASS 5:CLASS 6:CLASS 7:CLASS 8:CLASS 9:CLASS 10:CLASS 11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 17:CLASS 18:CLASS 19:CLASS 20:CLASS

L37 STRUCTURE UPLOADED

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FULL SUBSET SEARCH INITIATED 17:39:33 FILE 'REGISTRY'
FULL SUBSET SCREEN SEARCH COMPLETED - 19567 TO ITERATE

100.0% PROCESSED 19567 ITERATIONS 603 ANSWERS
SEARCH TIME: 00.00.01

L38 603 SEA SUB=L5 SSS FUL L37

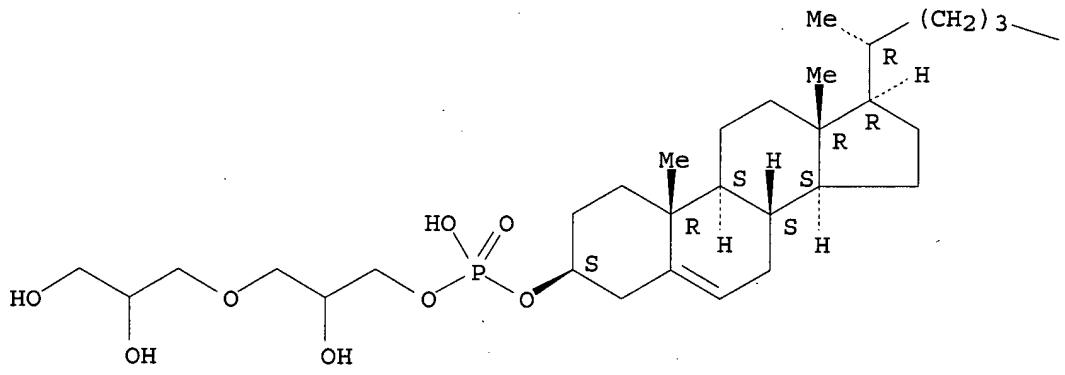
=> d l38 scan

L38 603 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Cholest-5-en-3-ol (3β)-, 3-(2,3-dihydroxypropoxy)-2-hydroxypropyl

hydrogen phosphate (9CI)
MF C33 H59 O8 P
CI COM

Absolute stereochemistry.

PAGE 1-A



PAGE 1-B

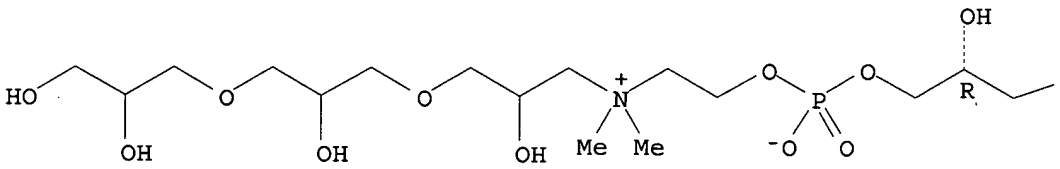
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):3

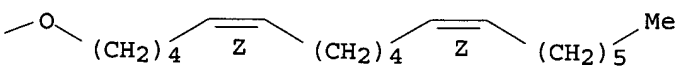
L38 603 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN 3,5,9-Trioxa-4-phosphaheptacosa-14,20-dien-1-aminium, N-[3-[3-(2,3-dihydroxypropoxy)-2-hydroxypropoxy]-2-hydroxypropyl]-4,7-dihydroxy-N,N-dimethyl-, inner salt, 4-oxide, (7R,14Z,20Z)- (9CI)
MF C34 H68 N O12 P

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



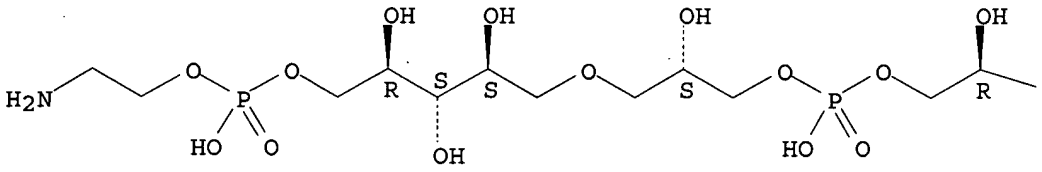
PAGE 1-B



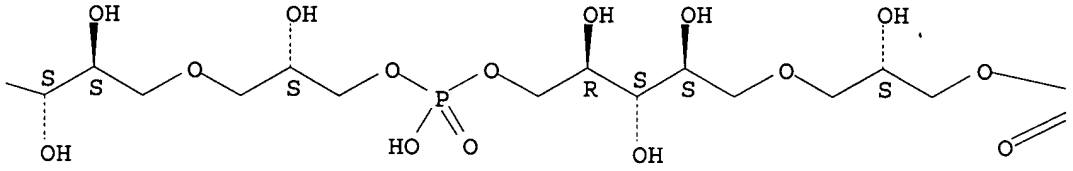
L38 603 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN D-Ribitol, O-5-O-[(2-aminoethoxy)hydroxyphosphinyl]-D-ribitol-1-O-yl[(2S)-2-hydroxy-1,3-propanediyl]oxyphosphinico-(1→5)-O-D-ribitol-1-O-yl[(2S)-2-hydroxy-1,3-propanediyl]oxyphosphinico-(1→5)-O-D-ribitol-1-O-yl[(2S)-2-hydroxy-1,3-propanediyl]oxyphosphinico-(1→5)-O-D-ribitol-1-O-yl[(2S)-2-hydroxy-1,3-propanediyl]oxyphosphinico-(1→5)-1-O-[(2S)-2,3-dihydroxypropyl]- (9CI)
MF C42 H92 N O46 P5

Absolute stereochemistry.

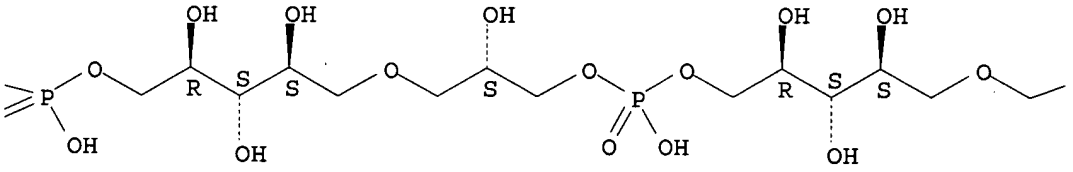
PAGE 1-A



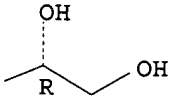
PAGE 1-B



PAGE 1-C

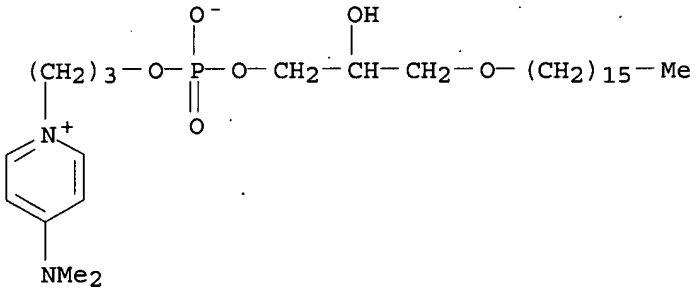


PAGE 1-D



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L38 603 ANSWERS REGISTRY COPYRIGHT 2006 ACS on STN
IN Pyridinium, 1-(5,8-dihydroxy-5-oxido-4,6,10-trioxa-5-phosphahexacos-1-yl)-4-(dimethylamino)-, inner salt (9CI)
MF C29 H55 N2 O6 P



HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	40.28	936.93

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-20.25

FILE 'CAPLUS' ENTERED AT 17:40:04 ON 11 DEC 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 11 Dec 2006 VOL 145 ISS 25
FILE LAST UPDATED: 10 Dec 2006 (20061210/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l38
L39 552 L38

=> s l39 and (restenosis or neointima)
7958 RESTENOSIS
1737 NEOINTIMA
L40 2 L39 AND (RESTENOSIS OR NEOINTIMA)

=> d l40 1-2 ti abs bib

L40 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation
AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPARγ, abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPARγ. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPARγ or antagonists of PPARγ that inhibit PPARγ signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.
AN 2004:857161 CAPLUS <<LOGINID::20061211>>
DN 141:343506
TI Lysophosphatidic acid analogs and inhibition of neointima formation
IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA USA
SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004204383	A1	20041014	US 2004-821739	20040409
	AU 2004229467	A1	20041028	AU 2004-229467	20040409
	CA 2521189	AA	20041028	CA 2004-2521189	20040409
	WO 2004091496	A2	20041028	WO 2004-US11016	20040409
	WO 2004091496	A3	20050324		
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	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1613298	A2	20060111	EP 2004-759365	20040409
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
PRAI	US 2003-462274P	P	20030411		
	WO 2004-US11016	W	20040409		

L40 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AB Neointimal lesions are characterized by accumulation of cells within the arterial wall and are a prelude to atherosclerotic disease. Here the authors report that a brief exposure to either alkyl ether analogs of the growth factor-like phospholipid lysophosphatidic acid (LPA), products generated during the oxidative modification of low d. lipoprotein, or to unsatd. acyl forms of LPA induce progressive formation of neointima in vivo in a rat carotid artery model. This effect is completely inhibited by the peroxisome proliferator-activated receptor

(PPAR)γ antagonist GW9662 and mimicked by PPARγ agonists Rosiglitazone and 1-O-hexadecyl-2-azeleoylphosphatidylcholine. In contrast, stearoyloxovalerylphosphatidylcholine, a PPARα agonist and the polypeptides epidermal growth factor, platelet-derived growth factor, and vascular endothelial growth factor failed to elicit neointima . The structure-activity relation for neointima induction by LPA analogs in vivo is identical to that of PPARγ activation in vitro and disparate from that of LPA G protein-coupled receptor activation. Neointima-inducing LPA analogs up-regulated the CD36 scavenger receptor in vitro and in vivo and elicited dedifferentiation of cultured vascular smooth muscle cells that was prevented by GW9662. These results suggest that selected LPA analogs are important novel endogenous PPARγ ligands capable of mediating vascular remodeling and that activation of the nuclear transcription factor PPARγ is both necessary and sufficient for neointima formation by components of oxidized low d. lipoprotein.

AN 2004:242383 CAPLUS <<LOGINID::20061211>>
DN 140:373126
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AU Zhang, Chunxiang; Baker, Daniel L.; Yasuda, Satoshi; Makarova, Natalia; Balazs, Louisa; Johnson, Leonard R.; Marathe, Gopal K.; McIntyre, Thomas M.; Xu, Yong; Prestwich, Glenn D.; Byun, Hoe-Sup; Bittman, Robert; Tigyi, Gabor
CS Vascular Biology Center of Excellence, The University of Tennessee Health Science Center, Memphis, TN, 38163, USA
SO Journal of Experimental Medicine (2004), 199(6), 763-774
CODEN: JEMEAV; ISSN: 0022-1007
PB Rockefeller University Press
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l39 and atherosclerosis
51957 ATHEROSCLEROSIS
L41 6 L39 AND ATHEROSCLEROSIS

=> d l41 1-6 ti

L41 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation

L41 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI The plaque lipid lysophosphatidic acid stimulates platelet activation and platelet-monocyte aggregate formation in whole blood: involvement of P2Y1 and P2Y12 receptors

L41 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid induces neointima formation through PPARγ activation

L41 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Subtype-Selective Antagonists of Lysophosphatidic Acid Receptors Inhibit Platelet Activation Triggered by the Lipid Core of Atherosclerotic Plaques

L41 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Ligands for G protein coupled receptors and methods of using them

L41 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Effect of platelet activating factor on the kinetics of LDL oxidation in vitro

=> d l41 2 3 5 6 ti abs bib

L41 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI The plaque lipid lysophosphatidic acid stimulates platelet activation and platelet-monocyte aggregate formation in whole blood: involvement of P2Y1 and P2Y12 receptors
AB Despite the fact that lysophosphatidic acid (LPA) has been identified as a main platelet-activating lipid of mildly oxidized low-d. lipoprotein (LDL) and human atherosclerotic lesions, it remains unknown whether it is capable of activating platelets in blood. The authors found that LPA at concns. slightly above plasma levels induces platelet shape change, aggregation, and platelet-monocyte aggregate formation in blood. 1-Alkyl-LPA (16:0 fatty acid) was almost 20-fold more potent than 1-acyl-LPA (16:0). LPA directly induced platelet shape change in blood and platelet-rich plasma obtained from all blood donors. However, LPA-stimulated platelet aggregation in blood was donor dependent. It could be completely blocked by apyrase and antagonists of the platelet ADP receptors P2Y1 and P2Y12. These substances also inhibited LPA-induced aggregation of platelet-rich plasma and aggregation and serotonin secretion of washed platelets. These results indicate a central role for ADP-mediated P2Y1 and P2Y12 receptor activation in supporting LPA-induced platelet aggregation. Platelet aggregation and platelet-monocyte aggregate formation stimulated by LPA was insensitive to inhibition by aspirin. The authors conclude that LPA at concns. approaching those found in vivo can induce platelet shape change, aggregation, and platelet-monocyte aggregate formation in whole blood and suggest that antagonists of platelet P2Y1 and P2Y12 receptors might be useful preventing LPA-elicited thrombus formation in patients with cardiovascular diseases.
AN 2004:302708 CAPLUS <<LOGINID::20061211>>
DN 140:421804
TI The plaque lipid lysophosphatidic acid stimulates platelet activation and platelet-monocyte aggregate formation in whole blood: involvement of P2Y1 and P2Y12 receptors
AU Haseruck, Nadine; Erl, Wolfgang; Pandey, Dharmendra; Tigyi, Gabor; Ohlmann, Philippe; Ravanat, Catherine; Gachet, Christian; Siess, Wolfgang
CS Institute for Prevention of Cardiovascular Diseases, University of Munich, Munich, D 80336, Germany
SO Blood (2004), 103(7), 2585-2592
CODEN: BLOOAW; ISSN: 0006-4971
PB American Society of Hematology
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L41 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AB Neointimal lesions are characterized by accumulation of cells within the arterial wall and are a prelude to atherosclerotic disease. Here the authors report that a brief exposure to either alkyl ether analogs of the growth factor-like phospholipid lysophosphatidic acid (LPA), products generated during the oxidative modification of low d. lipoprotein, or to unsatd. acyl forms of LPA induce progressive formation of neointima in vivo in a rat carotid artery model. This effect is completely inhibited by the peroxisome proliferator-activated receptor (PPAR)γ antagonist GW9662 and mimicked by PPARγ agonists Rosiglitazone and 1-O-hexadecyl-2-azeleoylphosphatidylcholine. In contrast, stearoyloxovalerylphosphatidylcholine, a PPARα agonist and the polypeptides epidermal growth factor, platelet-derived growth factor, and vascular endothelial growth factor failed to elicit neointima. The structure-activity relation for neointima induction by LPA analogs in vivo

is identical to that of PPARγ activation in vitro and disparate from that of LPA G protein-coupled receptor activation. Neointima-inducing LPA analogs up-regulated the CD36 scavenger receptor in vitro and in vivo and elicited dedifferentiation of cultured vascular smooth muscle cells that was prevented by GW9662. These results suggest that selected LPA analogs are important novel endogenous PPARγ ligands capable of mediating vascular remodeling and that activation of the nuclear transcription factor PPARγ is both necessary and sufficient for neointima formation by components of oxidized low d. lipoprotein.

AN 2004:242383 CAPLUS <<LOGINID::20061211>>
DN 140:373126
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AU Zhang, Chunxiang; Baker, Daniel L.; Yasuda, Satoshi; Makarova, Natalia; Balazs, Louisa; Johnson, Leonard R.; Marathe, Gopal K.; McIntyre, Thomas M.; Xu, Yong; Prestwich, Glenn D.; Byun, Hoe-Sup; Bittman, Robert; Tigyi, Gabor
CS Vascular Biology Center of Excellence, The University of Tennessee Health Science Center, Memphis, TN, 38163, USA
SO Journal of Experimental Medicine (2004), 199(6), 763-774
CODEN: JEMEAV; ISSN: 0022-1007
PB Rockefeller University Press
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L41 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2006 ACS on STN
TI Ligands for G protein coupled receptors and methods of using them
AB The present invention is directed to assays for potential drugs which modulate sphingosylphosphorylcholine (SPC) and/or lysophosphatidylcholine (LPC) binding to G protein-coupled receptors (GPCRs), and, in particular, GPR4 and TDAG8, diagnostic assays for disease conditions associated with expression of the receptors and ligands, methods of causing cellular effects by modulating such binding, methods of treating disease conditions associated with SPC and/or LPC expression or GPCR expression, and synthetic peptide analogs which bind to GPCRs. Among examples provided are: GPR4 and TDAG8 expression and distribution in human tissues, effects of SPC and LPCs on transient increases in intracellular Ca2+ in GPR4-transfected MCF10A cells, binding of SPC and LPC to GPR4 and TDAG8, LPC and SPC activation of SRE reporter system in HEK293 cells, SPC and LPC activation of ERK MAP kinase, SPC stimulation of DNA synthesis, elevation of SPC levels in ovarian cancer, and identification of a peptide from a phage library which blocks SPC-stimulated growth inhibition in HEY cells.
AN 2002:240585 CAPLUS <<LOGINID::20061211>>
DN 136:273226
TI Ligands for G protein coupled receptors and methods of using them
IN Xu, Yan; Zhu, Kui
PA The Cleveland Clinic Foundation, USA
SO PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002024222	A2	20020328	WO 2001-US29446	20010920
	WO 2002024222	A3	20030807		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

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        RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG,
            KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR,
            IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
            GQ, GW, ML, MR, NE, SN, TD, TG
    US 2002107197        A1    20020808    US 2001-956636         20010920
PRAI US 2000-234249P     P     20000920

L41  ANSWER 6 OF 6  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Effect of platelet activating factor on the kinetics of LDL oxidation in
     vitro
AB   Oxidatively modified low-d.-lipoprotein (LDL) might contribute to the
     atherosclerotic process.  This study was performed to examine an effect of
     platelet-activating factor (PAF) and of synthetic PAF analogs on
     Cu(II)-induced oxidation of LDL in vitro: The D- and L-isomers of PAF and
     analogs with short-chain sn-2-substituents, 1-O-alkyl-2-butyryl-sn-glycero-
     3-phosphocholine and 1-O-alkyl-2-heptanoyl-sn-glycero-3-phosphocholine,
     were found to be the most effective inhibitors of LDL oxidation  Oxidation was
     inhibited completely at PAF concns. above 100 μM.  Lyso-PAF and analogs
     carrying longer chains at the sn-2 position were less effective.  These
     results thus provide evidence for the involvement of other parameters in
     LDL oxidation beyond the content of natural antioxidants like vitamin E and
     β-carotene.
AN   1993:252470  CAPLUS <<LOGINID::20061211>>
DN   118:252470
TI   Effect of platelet activating factor on the kinetics of LDL oxidation in
     vitro
AU   Deigner, Hans-Peter; Dresel, Hans Alois
CS   Pharm.-Chem. Inst., Univ. Heidelberg, Im Neuenheimer Feld 364, Heidelberg,
     D-6900, Germany
SO   FEBS Letters (1993), 317(3), 202-6
     CODEN: FEBLAL; ISSN: 0014-5793
DT   Journal
LA   English


=> d his

     (FILE 'HOME' ENTERED AT 16:52:30 ON 11 DEC 2006)

     FILE 'REGISTRY' ENTERED AT 16:52:43 ON 11 DEC 2006
L1             STRUCTURE UPLOADED
L2          50 S L1
L3             STRUCTURE UPLOADED
L4          50 S L3
L5       19567 S L3 SSS FULL

     FILE 'CAPLUS' ENTERED AT 16:55:16 ON 11 DEC 2006
L6        5385 S L5/THU
L7           3 S L6 AND NEOINTIMA
L8          70 S L6 AND ATHEROSCLEROSIS
L9          27 S L8 NOT PY>2004
L10          1 S L9 AND LYSOPHOSPHATIDIC
L11          0 S L9 AND (PPAR(W)GAMMA)

     FILE 'USPATFULL' ENTERED AT 17:00:27 ON 11 DEC 2006
L12       2960 S L5
L13         14 S L12 AND NEOINTIMA
L14          1 S L13 AND LYSOPHOSPHATIDIC
L15        334 S L12 AND ATHEROSCLEROSIS
L16        209 S L15 NOT PY>2004
L17         19 S L16 AND LYSOPHOSPHATIDIC

     FILE 'REGISTRY' ENTERED AT 17:03:45 ON 11 DEC 2006
L18            STRUCTURE UPLOADED
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L19           1 S L18 FAM FULL

     FILE 'CAPLUS' ENTERED AT 17:04:22 ON 11 DEC 2006
L20          53 S L19
L21           3 S L20 AND ATHEROSCLEROSIS
L22           1 S L20 AND NEOINTIMA
L23           0 S L20 AND STENT
L24          12 S L20 NOT PY>2003

     FILE 'REGISTRY' ENTERED AT 17:32:21 ON 11 DEC 2006
L25             STRUCTURE UPLOADED
L26         145 S L25  SUB=L5 FULL

     FILE 'CAPLUS' ENTERED AT 17:33:40 ON 11 DEC 2006
L27         111 S L26
L28           0 S L27 AND (RESTENOSIS OR NEOINTIMA)
L29           0 S L27 AND (PPAR(W)GAMMA)

     FILE 'REGISTRY' ENTERED AT 17:34:35 ON 11 DEC 2006
L30             STRUCTURE UPLOADED
L31          26 S L30  SUB=L5 FULL

     FILE 'CAPLUS' ENTERED AT 17:36:04 ON 11 DEC 2006
                S L30

     FILE 'REGISTRY' ENTERED AT 17:36:08 ON 11 DEC 2006
L32           1 S L30

     FILE 'CAPLUS' ENTERED AT 17:36:09 ON 11 DEC 2006
L33           3 S L32
L34          10 S L31
L35           7 S L34 NOT PY>2004

     FILE 'USPATFULL' ENTERED AT 17:37:31 ON 11 DEC 2006
L36          10 S L31

     FILE 'REGISTRY' ENTERED AT 17:39:09 ON 11 DEC 2006
L37             STRUCTURE UPLOADED
L38         603 S L37  SUB=L5 FULL

     FILE 'CAPLUS' ENTERED AT 17:40:04 ON 11 DEC 2006
L39         552 S L38
L40           2 S L39 AND (RESTENOSIS OR NEOINTIMA)
L41           6 S L39 AND ATHEROSCLEROSIS

=> s l6 and restenosis
          7958 RESTENOSIS
L42          45 L6 AND RESTENOSIS

=> s l42 not py>2004
       2477817 PY>2004
L43          25 L42 NOT PY>2004

=> d l43 1-25 ti

L43  ANSWER 1 OF 25  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Peptide and peptide analog apolipoprotein A-I agonists and their use to
     treat dyslipidemic disorders

L43  ANSWER 2 OF 25  CAPLUS  COPYRIGHT 2006 ACS on STN
TI   Intramural Delivery of Recombinant Apolipoprotein A-IMilano/Phospholipid
     Complex (ETC-216) Inhibits In-Stent Stenosis in Porcine Coronary Arteries

L43  ANSWER 3 OF 25  CAPLUS  COPYRIGHT 2006 ACS on STN
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TI Gene transfer of human vascular endothelial growth factor 165 for prevention of stent restenosis after transjugular intrahepatic portosystemic shunt

L43 ANSWER 4 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Therapy of proliferative disorders by direct irradiation of cell nuclei with tritiated nuclear targeting agents

L43 ANSWER 5 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Receptor antagonist-lipid conjugates and delivery vehicles containing same

L43 ANSWER 6 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Cationic lipid-mediated transfection of bovine aortic endothelial cells inhibits their attachment

L43 ANSWER 7 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Optimization of nonviral gene transfer of vascular smooth muscle cells in vitro and in vivo

L43 ANSWER 8 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Surface modification of liposomes for selective cell targeting in cardiovascular drug delivery

L43 ANSWER 9 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Focal arterial transgene expression after local gene delivery

L43 ANSWER 10 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Pharmaceutical composition in the form of a nucleic acid lipid complex, the production thereof and its use in gene therapy

L43 ANSWER 11 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Toxicity, uptake kinetics and efficacy of new transfection reagents: Increase of oligonucleotide uptake

L43 ANSWER 12 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Preparation of vitronectin receptor antagonist pharmaceuticals

L43 ANSWER 13 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Preparation of vitronectin receptor antagonist pharmaceuticals

L43 ANSWER 14 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Ribozyme therapy for the treatment and/or prevention of restenosis

L43 ANSWER 15 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Agent for gene therapy of tumors and neurodegenerative, cardiovascular, and autoimmune diseases

L43 ANSWER 16 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Ribozyme-mediated inhibition of cell proliferation: A model for identifying and refining a therapeutic ribozyme

L43 ANSWER 17 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI P-selectin translocation to vascular epithelial lumen by ionizing radiation, and therapeutic use

L43 ANSWER 18 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Arterial Uptake of Biodegradable Nanoparticles: Effect of Surface Modifications

L43 ANSWER 19 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Induction of E-selectin for targeting therapeutic agents to disease-associated vascular endothelial cells

L43 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN

TI Lipid constructs for targeting oligonucleotides to vascular smooth muscle tissue

L43 ANSWER 21 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lipid constructs for cytoplasmic delivery of antisense oligonucleotides

L43 ANSWER 22 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Antisense DNAs to cyclins and cyclin kinases for inhibition of proliferation of vascular smooth muscle cells

L43 ANSWER 23 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Surface-modified nanoparticles and method of making and using them

L43 ANSWER 24 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method for treating diseases mediated by cellular proliferation in response to PDGF, EGF, FGF and VEGF

L43 ANSWER 25 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Inhibition of proliferation of vascular smooth muscle cells by antisense oligonucleotides against cyclins and cyclin-dependent kinases

=> d l43 3 5 8 10 20 21 ti abs bib

L43 ANSWER 3 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Gene transfer of human vascular endothelial growth factor 165 for prevention of stent restenosis after transjugular intrahepatic portosystemic shunt
AB OBJECTIVE: To test the hypothesis that locally directed gene transfer of human vascular endothelial growth factor 165 (hVEGF165) is able to passivate hepatic venous metallic stents by accelerating endothelialization and augmenting the biocompatibility of endovascular stents. METHODS: Complexes of pAdtrackCMV-hVEGF165 and lipofectamine were smeared homogeneously on the surface of the stents coated with poly-L-lysine. Bare stents were used as controls. All stents were implanted into the right hepatic vein by transjugular intrahepatic venous stem deployment. RESULTS: At the end of the first week after implantation, green fluorescence and the expression of the hVEGF165 gene were detected in the transferred stent vessels of the treated group, but were not detected in the control group. SEM revealed that the endothelialization of stents was more pronounced in the treated group than that in the control group. At the end of the eighth week after implantation, quant. angiog. anal. showed the internal diameter of the stent was significantly greater in the treated group than in the control group. The de novo intima, their mean areas and percentage cross-sectional area narrowing in the treated group were significantly less than those in the control group. Immunohistochem. anal. indicated that the proliferation of vascular smooth muscle cells was more active in the control group than that in the treated group. CONCLUSION: Local gene transfer of hVEGF165 can passivate endovascular stents by accelerating stent endothelialization and enhancing their biocompatibility in the hepatic vein, resulting in a reduction of thrombus formation and attenuation of intimal hyperplasia.
AN 2003:208614 CAPLUS <<LOGINID::20061211>>
DN 139:1408
TI Gene transfer of human vascular endothelial growth factor 165 for prevention of stent restenosis after transjugular intrahepatic portosystemic shunt
AU Li, Zi Jun; Cheng, Xiao Ming; Hu, Ping Jin; Luo, Peng Fei; Qiao, Hui; Lin, Qiu Xiong; Wang, Qi Yi
CS Department of Gastroenterology, Guangdong Provincial People's Hospital, Guangzhou, Guangdong, Peop. Rep. China
SO Chinese Journal of Digestive Diseases (2002), 3(4), 159-163
CODEN: CJDDA9; ISSN: 1443-9611
PB Blackwell Publishing Asia Pty Ltd.

DT Journal
LA English
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L43 ANSWER 5 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Receptor antagonist-lipid conjugates and delivery vehicles containing same
AB Vesicular drug delivery vehicles, such as liposomes, comprise a targeting ligand which comprises a non-biol., biomimetic antagonist to a receptor that is upregulated at a disease site, directly or indirectly chemical linked to a polar head group of a vesicle-forming lipid. The non-biol., biomimetic antagonist is an antagonist to a receptor upregulated in the vascular endothelium of inflammation, infection or tumor sites, selected from integrin receptors, prostate specific membrane antigen (PSMA) receptor, herceptin, Tie 1 and Tie 2 receptors, ICAM1, folate receptor, bFGF receptor, EGF receptor, VEGF receptor, PDGF receptor, etc. The vesicle-forming lipid is selected from phospholipids, sterols, glycolipids, cationic lipids, sphingolipids, glycerolipids, hydrophilic polymer derivs. of these lipids, gemini surfactants, etc. For example, liposomes were prepared containing lipid conjugates with a vitronectin receptor antagonist, (S)-7-[[N--(4-aminobutyl)-N-(benzimidazol-2-yl-methyl)]amino]carbonyl-4-methyl-3-oxo-2,3,4,5-tetrahydro-1H-1,4-benzodiazepine-2-acetic acid (preparation given) 0.5 mol%, DSPC 54.5 mol%, and cholesterol 45 mol%. The liposomes were loaded with topotecan using ion gradient or polymer gradient loading/retaining techniques and administered to a patient diagnosed with ovarian cancer to inhibit growth of the cancerous tumor. A dosing regimen was 1.5 mg/m2 of the topotecan liposomes given as a 30 min infusion over the course of 1-3 days in a week for 2 wk in a 21 day cycle, repeated for 4 cycles.
AN 2002:353239 CAPLUS <<LOGINID::20061211>>
DN 136:374827
TI Receptor antagonist-lipid conjugates and delivery vehicles containing same
IN Ellens, Harma M.; Monck, Myrna A.; Yeh, Ping-Yang
PA Smithkline Beecham Corporation, USA
SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002036073	A2	20020510	WO 2001-US46206	20011029
	WO 2002036073	A3	20021205		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002025878	A5	20020515	AU 2002-25878	20011029
	EP 1341497	A2	20030910	EP 2001-992551	20011029
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004512345	T2	20040422	JP 2002-538885	20011029
	US 2004013720	A1	20040122	US 2003-415160	20030425
PRAI	US 2000-245140P	P	20001102		
	WO 2001-US46206	W	20011029		

OS MARPAT 136:374827

L43 ANSWER 8 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Surface modification of liposomes for selective cell targeting in

cardiovascular drug delivery
AB Cardiovascular disease processes such as atherosclerosis, restenosis, and inflammation are typically localized to discrete regions of the vasculature, affording great opportunity for targeted pharmacol. treatment. Liposomes are potentially advantageous targeted drug carriers for such intravascular applications. To facilitate their use as drug delivery vehicles, we have considered three components of liposome design: (i) identification of candidate cell surface receptors for targeting; (ii) identification of ligands that maintain binding specificity and affinity; and (iii) prevention of rapid nonspecific clearance of liposomes into the reticuloendothelial organs. In this report, we describe our work in developing liposomal surface modifications that address both targeting and clearance. An arginine-glycine-aspartic acid (RGD) containing peptide was used as a model ligand to target liposomes to the integrin GPIIb-IIIa on activated platelets. Addnl., oligodextran surfactants incorporated into liposomes provided insight into the effect of vesicle perturbations on liposome clearance, and the importance of mol. geometry in designing oligosaccharide surface modifications. Together these studies demonstrate the feasibility of using peptides to guide liposomes to desired receptors, and illustrate the influence of vesicle stability on liposome interactions in vivo. Furthermore, they underscore the importance of simultaneously considering both targeting specificity and vesicle longevity in the design of effective targeted drug delivery systems.
AN 2001:932150 CAPLUS <<LOGINID::20061211>>
DN 137:237522
TI Surface modification of liposomes for selective cell targeting in cardiovascular drug delivery
AU Lestini, Brian J.; Sagnella, Sharon M.; Xu, Zhong; Shive, Matthew S.; Richter, Nancy J.; Jayaseharan, Johnsamuel; Case, Aubrey J.; Kottke-Marchant, Kandice; Anderson, James M.; Marchant, Roger E.
CS Department of Biomedical Engineering, Case Western Reserve University, Cleveland, OH, 44106, USA
SO Journal of Controlled Release (2002), 78(1-3), 235-247
CODEN: JCREEC; ISSN: 0168-3659
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L43 ANSWER 10 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Pharmaceutical composition in the form of a nucleic acid lipid complex, the production thereof and its use in gene therapy
AB The invention relates to a pharmaceutical composition in the form of a nucleic acid lipid complex containing at least one cationic lipid, at least one non-cationic lipid, at least one nucleic acid that codes for a protein used for treating vascular diseases, especially a protein having vasodilatory and/or angiogenic properties, and optionally containing appropriate auxiliary and/or addition agents. According to the invention, the cationic lipid contains a group derived from cholesterol on which at least one cationic amino group selected from a primary, secondary, tertiary amino group and/or from a quaternary ammonium salt is bound via a connecting group selected from carboxamides and carbamoyls, and via a spacer comprised of a linear or branched alkyl group with 1 to 20 carbon atoms. In addition, the size of the nucleic acid lipid complexes ranges from approx. 300 to 800 nm. The invention also relates to the production of the pharmaceutical composition and to its use in gene therapy.
AN 2001:208132 CAPLUS <<LOGINID::20061211>>
DN 134:242634
TI Pharmaceutical composition in the form of a nucleic acid lipid complex, the production thereof and its use in gene therapy
IN Schletter, Jens

PA Cardiogene Gentherapeutische Systeme A.-G., Germany
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001019400	A2	20010322	WO 2000-EP8996	20000914
	WO 2001019400	A3	20020214		
	W: AU, CA, CN, CZ, HU, ID, IL, IN, JP, KR, NO, PL, RU, SG, SI, SK, TR, US, ZA				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	DE 19944262	A1	20010329	DE 1999-19944262	19990915
	EP 1216027	A2	20020626	EP 2000-967678	20000914
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, CY				
PRAI	DE 1999-19944262	A	19990915		
	WO 2000-EP8996	W	20000914		

L43 ANSWER 20 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lipid constructs for targeting oligonucleotides to vascular smooth muscle tissue
AB A lipid construct comprising an aminomannose-derivatized cholesterol suitable for targeting smooth muscle cells and tissue is claimed. Preferred formulations contain 6-(cholest-5-en-3β-yloxyl)hexyl-6-amino-6-deoxy-1-thio-α-D-mannopyranoside in liposome formulations wherein the formulations are delivered generally to arteries using percutaneous transluminal coronary angioplasty procedures. These formulations have applications in the reduction of restenosis.
AN 1996:607513 CAPLUS <<LOGINID::20061211>>
DN 125:257174
TI Lipid constructs for targeting oligonucleotides to vascular smooth muscle tissue
IN Male-Brunne, Roxanne
PA California Institute of Technology, USA
SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9624333	A1	19960815	WO 1996-US1807	19960209
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	US 5660855	A	19970826	US 1995-386579	19950210
	AU 9650220	A1	19960827	AU 1996-50220	19960209
PRAI	US 1995-386579	A	19950210		
	WO 1996-US1807	W	19960209		

L43 ANSWER 21 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
TI Lipid constructs for cytoplasmic delivery of antisense oligonucleotides
AB A lipid construct comprising an aminomannose derivatized cholesterol suitable for targeting smooth muscle cells and tissue is claimed. Preferred formulations contain 6-(cholest-5-en-3β-xyloxyl)hexyl-6-amino-6-deoxy-1-thio-α-D-mannopyranoside in liposome formulations wherein the formulations are delivered generally to arteries using

percutaneous transluminal coronary angioplasty procedures. These formulations have applications in the reduction of restenosis.

AN 1996:605480 CAPLUS <<LOGINID::20061211>>

DN 125:257172

TI Lipid constructs for cytoplasmic delivery of antisense oligonucleotides

IN Male-Brunne, Roxanne; Proffitt, Richard

PA Nexstar Pharmaceuticals, Inc., USA

SO PCT Int. Appl., 48 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9624334	A1	19960815	WO 1996-US1960	19960208
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9649236	A1	19960827	AU 1996-49236	19960208
PRAI	US 1995-386577	A	19950210		
	WO 1996-US1960	W	19960208		